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# PLASMA LACTATE AND PYRUVATE RESPONSE TO EXERCISE AND TRAINING

by

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled " Plasma Lactate and Pyruvate Response to Exercise and Training " submitted by Raymond Einar Lovlin in partial fulfilment of the requirements for the degree of Master of Science.

# ABSTRACT

Eighteen male University of Alberta students were investigated to determine the effect of training on the plasma concentrations of lactate and pyruvate. Three groups were compared under maximal and submaximal exercise conditions. These were highly trained, semi-trained, and sedentary groups. Arterial and venous blood samples were taken at rest, during the exercise session, at the point of exhaustion and during the recovery period. The samples were analyzed for plasma concentrations of lactate and pyruvate. Comparisons were made between groups as well as within groups for possible differences in arterial and venous samples.

There was no significant difference between the three groups with respect to plasma lactate concentrations. However, the plasma lactate response of the working limb during maximal exercise was significantly different from the non-working limb in the trained and sedentary groups. Resting pyruvate concentrations were significantly different between the three groups. During the recovery period lactate uptake by the working muscle was evident suggesting that local adaptations had occured within the trained muscle.

Arterial and venous concentrations of plasma lactate and pyruvate were almost identical during submaximal exercise. The consistently greater pyruvate concentrations in the

trained group suggests that training resulted in an adaptive response within the muscle or perhaps the blood itself.

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# CHAPTER I

## STATEMENT OF THE PROBLEM

### Introduction

The acute responses that occur in organs and tissues due to the energy demands of exercise have attracted considerable interest for many years. In efforts to elucidate the mechanisms of energy production, research has concentrated on glycolysis, glycogenolysis, the Kreb's Cycle, oxidative phosphorylation, the mobilization of FFA*, and more recently, mitochondrial alterations ( 1,33,38,39,40,79,81 ). In most instances, it is implied that the efficiency of the various pathways, and consequently the production of energy is related to the availability of oxygen to the metabolizing system. If oxygen is readily available, the system functions to optimum capacity with little metabolite accumulation. If adequate oxygen is not readily available however, as during altitude exposure, anemia, certain cardio-respiratory ailments, or exercise, the capacity of the system to produce energy is reduced and the subsequent production of intermediate metabolites increased.

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*Abreviations used in this thesis are the following: FFA=Free Fatty Acids; NAD=nicotinamide adenine dinucleotide; NADH=reduced NAD; ATP=adenosine triphosphate; ADP=adenosine diphosphate; H=hydrogen; $PO_4$=phosphate; Co-A=coenzyme A; $CO_2$=carbon dioxide; $H_2O$=water; $O_2$=oxygen; OH=hydroxyl radical; $MVO_2$=maximum oxygen uptake; 2,3-DPG=2,3-diphosphoglycerate.

This increase in metabolite production, particularly evident during exercise, is often interpreted as being reflective of the physiological state of the individual ( 70 ). The inference here of course is, that the individual who has attained a certain level of physical conditioning will adapt more efficiently to imposed demands than the unconditioned individual. The assumption is made that the trained body supplies and utilizes oxygen more efficiently than the untrained body. Although there have been numerous attempts to relate the production and removal of lactic acid to the physiological state of the individual, the evidence certainly is not conclusive that such a relationship exists. It is the intent of this investigation to provide evidence which may assist in clarifying or refuting some of the currently held concepts regarding the effects of training on plasma lactate and pyruvate accumulation during exercise.

## Statement of the Problem

The purpose of this investigation was to note the effects of training on the plasma concentrations of lactate and pyruvate. Arterial and venous blood samples were obtained from subjects as they exercised to exhaustion at both maximal and submaximal efforts. The samples were then analyzed for concentrations of lactate and pyruvate. Since in the past, adequate consideration has not been given to comparing metabolite variations between working and nonworking tissues, special attention was given to this aspect of the study.

Of particular interest, was a comparison in the plasma concentrations of lactate and pyruvate between highly trained and untrained individuals.

Rationale Behind the Study

Although there is general agreement in the literature with respect to lactate produced during maximal efforts, there is still considerable controversy about what actually occurs during submaximal efforts. A major point of contention appears to be whether in fact lactate production occurs during submaximal exercise.

Lactate and pyruvate production are apparently related to the availability of oxygen to the metabolizing tissue ( 33, 81 ). With the conversion of glucose or glycogen to lactate, a limited amount of energy in the form of ATP is provided when the oxygen supply is deficient. This is typically referred to as the anaerobic pathway for energy production and enables a tissue to maintain its function to a certain degree despite hypoxic conditions ( 33,81 ). Although controversy exists, it is assumed that the deficiency of oxygen causes an accumulation of reduced substrates necessitating an increase in lactic acid. Since lactate accumulation is indirectly related to oxygen supply, it seems pertinent to relate metabolite production to maximum oxygen uptake. In the past, this has not been emphasized; rather, work loads have been indiscriminately assigned with little regard for the subjects $\dot{M}VO_2$. In addition, it is becoming evident, that with pro-

longed conditioning, there are definite changes in mitochondrial structure and composition. Recently, evidence has been produced which illustrates a definite increase in number and enzymatic composition of trained skeletal muscle mitochondria ( 14,32,38,39,40 ). One would expect such alterations to influence metabolite accumulation.

With the aforesaid concepts receiving due consideration, perhaps a more valid assessment of the lactic acid mechanism and exercise is possible.

# CHAPTER II

# REVIEW OF THE LITERATURE

## Aerobic and Anaerobic Production of Energy

Glycolysis ( figure 1 ), the citric acid cycle ( figure 2 ), and oxidative phosphorylation ( figure 3 ), are three important cellular mechanisms available for ATP production. The latter two occur in the mitochondria while glycolysis is a cytoplasmic reaction. Since oxygen is directly involved with proton activity in the mitochondria, this is typically referred to as aerobic energy production. In the cytoplasm, however, ATP production occurs independent of oxygen and consequently accounts for the anaerobic contribution towards the ATP pool. The net contribution from 1 mole of glucose is 38 moles of ATP. The reactions may be summarized as follows:

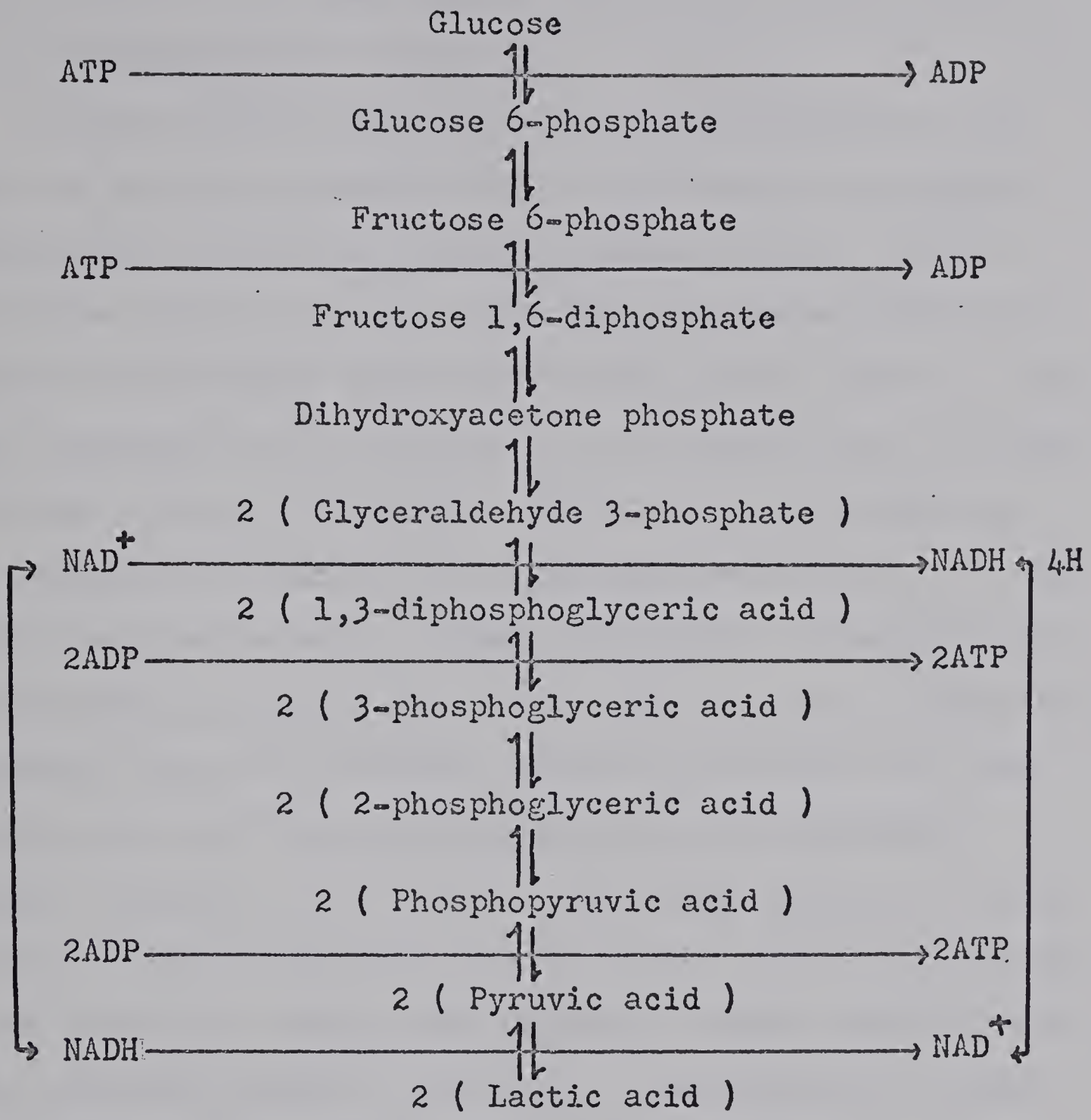
$$\text{Glucose} + 2\text{ADP} + PO_4 \longrightarrow 2 \text{ Pyruvic acid} + \underline{2\text{ATP}} + 4\text{H}$$

$$2 \text{ Acetyl CoA} + 6H_2O + 2\text{ADP} \longrightarrow 2CO_2 + \underline{2\text{ATP}} + 2\text{CoA} + 16\text{H}$$

The 20 hydrogens produced in the above reactions are shuttled through the cytochrome system ( figure 3 ) to contribute a total of 30 moles of ATP via the conversion

$$3P_i + 2\text{H} + \tfrac{1}{2}O_2 + 3\text{ADP} \longrightarrow H_2O + \underline{3\text{ATP}}.$$

The remaining 4 moles of ATP are accountable from the $H^+$ ions produced in the decarboxylation of pyruvic acid. The direct contribution from the anaerobic pathway accounts for two




Net reaction:

$$\text{Glucose} + 2\ \text{ADP} + 2\ PO_4 \dashrightarrow 2\ \text{Lactic acid} + 2\ \text{ATP} + 4H$$

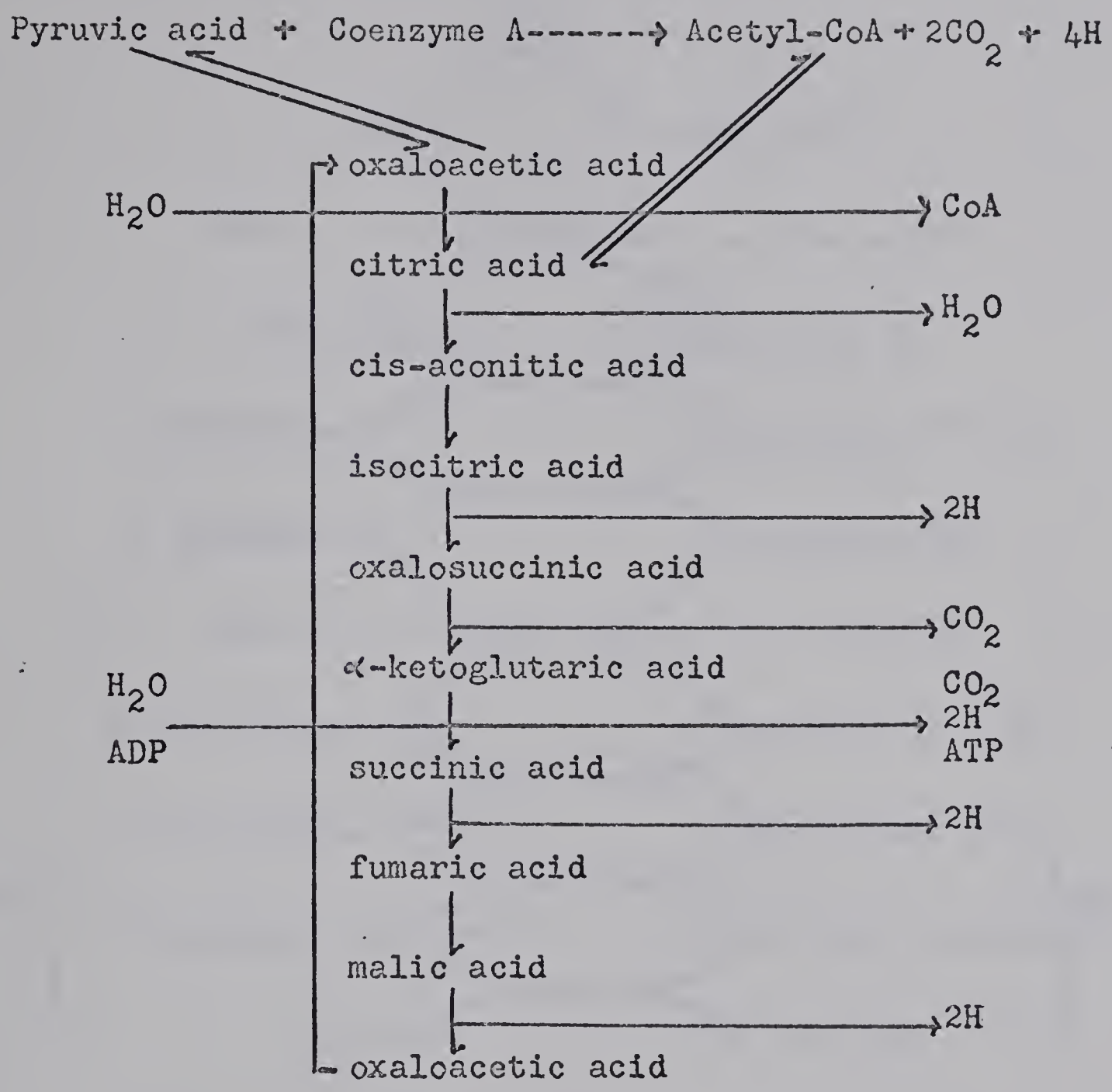
FIGURE 1

ANAEROBIC PRODUCTION OF ENERGY ( 33 )

moles of ATP. The remaining 36 moles result from oxidative phosphorylations ( 32,79 ).

During exercise and other stressful situations, the body may need more oxygen than it can immediately supply, consequently, proton accumulation often occurs. If the cytochrome system or other buffering mechanisms within the cellular environment cannot handle the proton buildup, there will ultimately be an increase in the concentration of NADH and other reduced substrates. This increase in NADH may also directly influence the lactate/pyruvate ratio. If the NADH is maintained above normal cytoplasmic concentrations, the anaerobic contribution of ATP will naturally be greater. Pyruvate, acting as a proton acceptor from NADH will form lactate plus $NAD^+$ and subsequently keep the glycolytic pathway reactive. As a result, the energy produced by glycolysis is often sufficient to maintain a certain degree of tissue activity, despite the hypoxia. During muscular activity anaerobic energy production is particularly evident along with a corresponding increase in lactate.

Aerobic energy production therefore depends upon oxygen availability while the anaerobic contribution is directly related to the cytoplasmic NAD/NADH ratio ( 51 ). During exercise, therefore, when the oxygen supply is often deficient, the production of ATP via oxidative phosphorylation is hindered and the anaerobic contribution is proportionately increased.

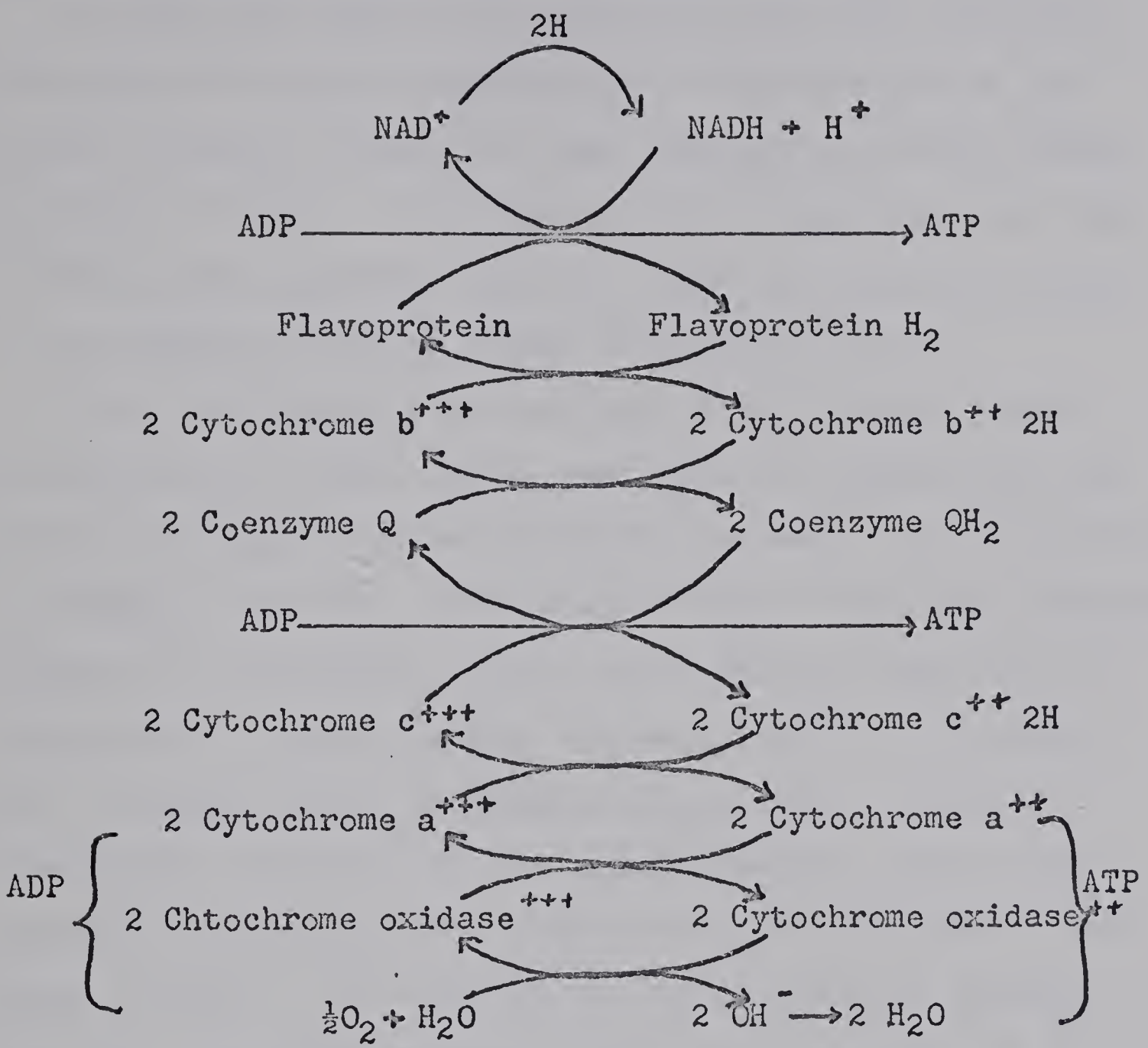



Net reaction:

$$2\ \text{Acetyl-CoA} + 6\ H_2O + 2\ \text{ADP} \longrightarrow 4\ CO_2 + 16H + 2\ \text{CoA} + 2\ \text{ATP}$$

FIGURE 2

THE CITRIC ACID CYCLE ( 33 )




Net reaction:

$$2H + \tfrac{1}{2}O_2 + 3\ ADP \dashrightarrow H_2O + 3\ ATP$$

FIGURE 3

AEROBIC ENERGY PRODUCTION ( 33 )

Lactic Acid, Oxygen, and Muscular Contraction

In the late 1800s, the implication was made, that the contraction of muscle depended upon the conversion of potential to active energy via some chemical mechanism within the muscle itself. In addition, it was suggested, that the amount of work performed could be taken as a direct measure of the chemical activity within the muscle ( 54 ).

The investigations at the turn of the century marked the beginning of many studies that were to indicate the importance of oxygen to the contractile process. It was found, for example, that the accumulation of one of the most evident products of contraction, lactic acid, depended upon the availability of oxygen to that tissue ( 27,28 ). By exposing a fatigued muscle to oxygen, investigators observed a considerable reduction in the lactate content, while under inadequate oxygenation the lactate content was found to increase ( 28,69 ). In addition to the circulatory system, therefore, these observations led to the suggestion of a local mechanism for lactate removal. Although this mechanism of removal was not understood ( 27,28,66 ), it was suggested that the disappearance of the lactate during recovery from fatigue did not only involve its oxidative removal in the form of carbonic acid and water, but indeed, suggested a reconstructive process to its chemical predecessor ( 27 ). Later, additional evidence was presented which indicated the importance of oxygen to the contractile process ( 66,69 ). Not only was a greater amount of oxygen

consumed during contraction, but, this increased consumption was found to persist long after the muscle had ceased to contract ( 15,16,27,28,41,66,69 ). The conclusion was drawn that oxygen was essential to the recovery process of exercise.

The first concerted effort at rationalizing the excess oxygen consumption following exercise was made in the mid-twenties when the concept of " oxygen debt " was formulated. Prolonged periods of recovery were attributed to excessive lactate accumulation and its subsequent conversion to glycogen ( 43 ). Since this formative period, considerable effort has gone into resolving the lactic acid-oxygen debt relationship ( 25,26,31,48,49,57,60 ), if indeed such a definitive relationship exists ( 1,47,48,51 ).

Margaria et al ( 57 ), attempted to indicate additional mechanisms that might be involved in payment of the oxygen debt resulting from exercise other than the lactate to glycogen conversion alone. These investigators suggested that the slowness of removal of lactate from the system resulted from the slowness of the process of its oxidation ( 57,59 ).

In efforts to clarify their theory, a distinction was made between an " alactacid oxygen debt " and a " lactacid oxygen debt "; the former occurs during the initial anaerobic conditions of exertion while the latter phase develops during later hypoxia if such occurs. The distinction is, that during the initial stages of exercise the immediate supply of energy is through sources other than those in-

volving production of appreciable lactic acid. During this phase, there is also a measureable oxygen debt developed. Since this initial oxygen debt cannot be attributed to the lactic acid mechanism, it has been labelled " alactacid ". That portion of the oxygen debt attributable to lactic acid develops when the source of energy is via glycolysis and is evident only when the work intensity exceeds a critical threshold.

Whereas in the past total lactate accumulation and oxygen debt were equated, Margaria et al ( 57 ) were able to accumulate an oxygen debt without any traceable differences in lactate production. In efforts to account for the alactacid debt, Margaria suggested possible replenishment of myoglobin stores and/or unknown anaerobic metabolites that are rapidly resynthesized during aerobic conditions ( 57,58,59 ). The implication is, that if the body is able to adapt and supply sufficient oxygen for the work at hand, the alactacid debt initially developed may in fact be simultaneously repayed.

During the 19-twenties and 19-thirties therefore, the theory was developed that lactic acid was a product of contraction, notably during anaerobic conditions. A considerable portion of the extra oxygen consumed during recovery from exercise was attributed to paying a portion of the oxygen debt which had resulted from lactate accumulation.

Recently, Huckabee ( 45 ) was able to demonstrate the accumulation of lactate in non-hypoxic conditions. Pre-

viously, lactate accumulation was directly associated with tissue hypoxia. Since the lactate content could vary in non-hypoxic conditions ( i.e., by infusion of sodium bicarbonate, pyruvate, or hyperventilation ), Huckabee suggested that the total lactate change was not a valid measurement of oxygen debt. His argument basically was, that if lactate bears a constant relationship to pyruvate so that normal oxidation-reduction reactions occur, there need not be an oxygen debt. If " excess lactate " developed such that normal redox reactions altered, Huckabee attributed this excess lactate to tissue hypoxia. Furthermore, since tissue hypoxia was very evident during exercise, and directly related to work intensity, Huckabee hypothesized a linear relationship between his concept of " excess lactate " and exertion. This interpretation eliminated the possibility of an alactic phase during exercise, i.e., excess lactate accumulated in accordance with the degree of tissue hypoxia. Previously, the implication was that a certain threshold in terms of work intensity had to be reached in order for a change in lactate to be apparent.

Since Huckabee's theories on the oxygen debt-lactic acid phenomenon, there have been numerous attempts at resolving the differences between his concept of excess lactate and Margaria's original threshold interpretation ( 49, 56,58,62,74,76,78 ). Considerable criticism has come from Keul ( 47,48 ), Alpert ( 1 ), and Olsen ( 62 ) who question the entire lactic acid-oxygen debt relationship. In addi-

tion, evidence has been presented which indicates that the NAD/NADH pools in the cell may in fact function independently ( 1,51,71 ). The mitochondrial membrane has been shown to be impermeable to NADH ( 71 ). Such independent functioning would provide a basis for questioning the established theories. Lactate production and utilization might then depend on mitochondrial or enzymatic efficiencies of the cell which only recently ( 14,32,38,39,40 ) have received considerable attention.

## Lactate Production and Exercise

### Maximal Efforts

Following exhaustive exercise at maximal efforts, most researchers agree that peak lactate values are not reached until five to ten minutes into the recovery period ( 17,20, 59 ). The delayed rise is attributed to the diffusion of lactate from the muscles into the plasma. DI Prampero *et al* ( 22 ) however, found tissue and blood lactic acid concentration " roughly " equal. Diamant *et al* ( 20 ), on the other hand, found muscle tissue lactate considerably greater than blood concentrations, at rest, as well as following exhaustive exercise.

One would expect, that with rapid diffusability of the lactate and subsequent circulation, an equilibrium between working and nonworking tissues might be attained. Williams ( 82 ) suggests that in order for the venous blood lactate concentration of the inactive arm muscles to be altered,

the legs must be doing very strenuous work.

According to Margaria ( 56,58 ), the only energy source available after 40 seconds of maximal efforts is via oxidative processes. The implication must be that lactate production reaches a maximum that is perhaps maintained throughout maximal efforts.

Submaximal Efforts

There is considerable controversy as to whether appreciable lactate production occurs during submaximal efforts. Saiki et al ( 70 ) contends that if lactate is produced during the alactic phase of exertion, this will disappear during later aerobic conditions. As long as the effort is below one's $\dot{M}VO_2$, Saiki states that lactate production will be curtailed since adequate oxygen is being supplied. Others ( 9,49,78 ) tend to support this hypothesis.

Substantial evidence is also available, however, to suggest continued lactate production below $\dot{M}VO_2$ efforts ( 19, 82 ).

Lactate Removal During Exercise

The heart ( 15,16,37,50 ), skeletal muscle ( 17,74 ), liver ( 68 ) and kidneys ( 52,53 ) are able to remove lactate from the blood during exercise. Rowell ( 68 ) estimates that approximately one half of the lactate produced during exercise is removed by the liver via gluconeogenesis. The myocardium, with an abundance of mitochondria, removes lac-

tate continuously regardless of the workload. Stainsby ( 74 ) provided evidence to indicate appreciable lactate uptake by skeletal muscle. Of particular interest, Stainsby noted that oxygen uptake appeared to be limited when the frequency of contractions increased beyond a critical value.

The Effect of Training on Lactate Production

Tipton ( 76 ) found no significant differences between trained and untrained rats with respect to respiratory rates of skeletal muscle, liver, diaphragm, and splanchnic tissues. He suggested that the enhanced capabilities of the trained tissue to respond to stress ( exercise ) must result from alterations in metabolic pathways or enzymatic efficiencies. The majority of the research available, however , points to decreased lactate production in the trained ( 9,70 ), if one can assume athletes are in good condition. Barnard ( 9 ) and Saiki ( 70 ) found lactate accumulation decreased as a result of training ( workloads were variable ). Williams ( 82 ), after an intensive training program indicated that with workloads of less than 800 kpm, there was no increase in lactate over resting values. Higher workloads, however, showed a definite increase over resting concentrations. Barnard ( 9 ) would tend to agree with Williams in that he suggested training would alter metabolic events such that there is greater aerobic capacity under submaximal conditions. According to Saiki ( 70 ), the production and removal of lactate seems to proceed faster in the athlete.

# CHAPTER III

## METHODS AND PROCEDURES

Eighteen male subjects ( 18 to 24 years of age ) from the University of Alberta, Edmonton were investigated. They were classified according to physical condition; six members from the University Wrestling Team as trained, six members from a physical education ( swimming class ) as semi-trained, and six sedentary members. The sedentary group which served as the control was inactive other than normal walking habits to and from classes.

The series of tests consisted of three sessions on a bi-cycle ergometer; an initial work capacity determination, followed by maximal and submaximal sessions to exhaustion. Blood samples were taken at regular intervals during the maximal and submaximal trials, prepared for lactate and pyruvate analysis and immediately frozen.

### Work Capacity Determinations

A predicted $\dot{M}VO_2$ was determined for all subjects as outlined by Astrand ( 3, 4, 5, 6 ). On two separate occasions subjects were assigned a workload by the examiner. In all trials, this workload resulted in a heart rate of 155±15 beats per minute as recorded by a Sanborn 500 Electrocardiogram. All subjects, on both occasions bicycled for at least 10 minutes i.e., until the heart rate was constant for a 4-5 minute

period. After appropriate transference procedures to the Astrand-Rhyming nomogram, the larger workload with respect to $\dot{M}VO_2$ prediction from the two trials was utilized as the initial resistance in the maximal trial. The submaximal workload ( 70% ) was determined by interpolation as employed by Astrand and Rhyming ( 3, 4 ).

Maximal and Submaximal Tests

Maximal Trials

Exercise was performed in a sitting position on a Monark bicycle ergometer and consisted of a series of workloads with uninterrupted pedalling. The pedal speed was maintained at 50 rpm with a metronone assisting. The initial resistance, which was constant for 1 minute, corresponded to the workload used to predict the subjects $\dot{M}VO_2$. This workload was then increased stepwise every minute by one-third of the original until the subject was unable to maintain the pace. By maintaining the 50 rpm throughout the testing session, 100% oxygen uptake was guaranteed. This procedure exhausted all subjects in three minutes or less, with termination of the exercise determined by the subjects inability to maintain the pace. Blood samples were taken before, immediately after, and 10 minutes after exercise.

Submaximal Trials

Exercise was performed per maximal trials, however, in this instance the subject pedalled at his predetermined work-

load which generated an estimated oxygen uptake of 70% of his $\dot{M}VO_2$. Again, termination was failure of the subject to maintain the pace. In this test, blood samples were taken before, at successive twenty minute intervals, immediately after, and 10 minutes after exercise to fatigue.

In both maximal and submaximal sessions, teflon catheters ( Venocarth - 16, 18-G bore, 11-½ Intravenous - Abbott Laboratories ) were inserted into the femoral vein and brachial artery following local anesthesia. All resting samples ( 5 mls ) were obtained after 1 hour of rest with the subject in the supine position with samples drawn simultaneously from both catheters. Exercise samples were similarly drawn with the subject on the bicycle ergometer.

Processing of Blood Samples

Precipitation of the samples with 6% perchloric acid ( 5 mls/5 mls blood ) was performed within 30 seconds of withdrawing the samples. Vigorous shaking in chilled centrifuge tubes for 10-15 seconds preceded centrifugation at 3,000 rpm for 5-10 minutes. If the filtrate was not clear, a second centrifugation was performed. Approximately 4 mls of the supernatant was transferred to a culture tube which was immediately placed in a solution of dry ice and 95% ethanol. The frozen samples were refrigerated for later lactate and pyruvate plasma determinations.

Analysis

Lactic and pyruvic acid concentrations were determined by the enzymatic method of the Sigma Chemical Company ( 72, 73 ).

Lactic Acid

Preparation of test and blank tubes proceeded as follows: An appropriate amount of the reducing mixture ( containing $NAD^{+}$, glycine buffer, $Mg^{++}$, and lactic dehydrogenase ) was placed in both test tubes. The thawed supernatant ( .10 mls protein free ) was added to the test tube while .10 mls of water were mixed with the contents of the blank tube. After gentle mixing, both tubes were incubated at 37°C for 1 hour. An O.D. recording was made on the test tube at 340 mu using the blank as reference. Calculations to obtain results in mg lactic acid per 100 mls of blood were made as outlined by the Sigma Chemical Company ( 72 ).

Pyruvic Acid

Three mls of the clear supernatant ( thawed ) and 1 ml of phosphate buffer ( stock 725-2 ) were mixed and placed in an ice bath for approximately 10 minutes. This procedure resulted in precipitation of potassium perchlorate crystals leaving 2 mls of supernatant. Test and blank tubes were prepared as follows: Phosphate buffer and $\alpha$-NADH were mixed with 2 mls of the supernatant previously obtained to form the test cuvette. The blank contained 2.9 mls of phosphate buffer ( stock no. 410-3 ). O.D. recordings were made on the test using the blank as reference.

To each of the cuvettes, .10 mls of lactic dehydrogenase solution ( E ) was added and thoroughly mixed. After waiting approximately 3 minutes, minimum $OD_{340}$ readings were made with the blank as reference. Calculations were determined to yield results in mg pyruvate/ 100 mls blood ( 73 ).

Statistical Analysis

An analysis of variance design with repeated measures on two factors was selected to determine whether production of lactate and pyruvate during maximal and submaximal exercise occured independent of the " fitness " level of the individual ( 83 ). Comparisons were made within groups as well as between groups. An alpha of 0.05 was considered appropriate throughout with Tukey's w applied when necessary. Submaximal data were treated graphically.

# CHAPTER IV

# RESULTS

## Symbols, Table Values and Definitions

The following are necessary for interpretation of the material in this chapter.

### Symbols

mg% .....units of concentration in all tables and graphs.
n .....refers to number of subjects.
a .....indicates significant differences between means.
AB .....refers to training X activity interaction.
AC .....refers to training X period interaction.
BC .....refers to activity X period interaction.
w .....refers to Tukey's w.

### Table Values

Table values represent the means ± the standard error of the mean.

Definitions

Lactate response is the change in the concentration of plasma lactate brought about by alterations in resting cellular metabolism.

Pyruvate response is the change in the concentration of plasma pyruvate brought about by alterations in resting cellular metabolism.

Exercise is of maximal and submaximal intensity such that exhaustion is reached in both instances. Oxygen uptake is maximal or below maximal depending on the severity of the exercise.

Exhaustion was determined by failure of the subject to maintain a specified pace.

Maximal exercise was of variable intensity producing exhaustion in less than three minutes.

Submaximal exercise was based on the subject's $M\dot{V}O_2$ and characterized by a considerably longer performance time.

Training level was determined by the degree to which the subjects participated in physical activity:

trained - University of Alberta wrestlers
semi-trained - Physical Education ( swimmers )
sedentary - Shuffleboard players

Activity refers to the working or nonworking state of the surrounding muscular tissue.

Period refers to the physiological state of the subject when the blood sample was obtained. During maximal and submaximal exercise, the Initial samples were obtained with

the subject in the resting state. The Final sample was obtained immediately following exhaustion produced by the exercise; the Recovery sample was drawn 10 minutes later. The submaximal 25%, 50%, and 75% values are mean plasma concentrations of lactate and pyruvate obtained by linear interpolation. Each individual profile was examined at points corresponding to 25%, 50%, and 75% of his total performance time. The lactate and pyruvate values indicated at these points were obtained and group means derived. A linear relationship was assumed.

Subject Reaction to Testing Procedures

Placement of the Teflon catheters in the brachial artery and femoral vein often resulted in mild discomfort for the subjects. This temporary discomfort, however, did not interfere with performance and none of the subjects reported subsequent ill effects.

Lactate and Pyruvate Response

The raw data for lactate and pyruvate response during maximal and submaximal exercise is contained in Appendix B. Results for the three way analysis of variance are shown in Table 1. Table 2 displays the anthropometric data as well as the results of the exercise tests. Tables 3 through 6 contain the means for lactate and pyruvate response during maximal exercise while Figures 4 through 7 are graphical representations of the same means.

Figures 8 through 11 portray the submaximal response with the 25%, 50%, and 75% values obtained by linear interpolation. The values from Table 11 were obtained by dividing the mean lactate concentration by the mean pyruvate concentration resulting in the lactate/pyruvate ratios. Figure 12 graphically displays these results.

Comparisons of the plasma lactate and pyruvate responses for working and nonworking limbs are graphically represented by Figures 13 through 18.

TABLE 1

SIGNIFICANT RESULTS FOR THE ANALYSIS OF VARIANCE

| Source of Variation | Maximal | | Submaximal | |
|---|---|---|---|---|
| | La | Py | La | Py |
| Groups (Trained vs Semi-trained vs Sedentary) | | * | * | * |
| Activity (Working vs Nonworking) | * ** | | | |
| Periods (Initial vs Final vs Recovery) | * ** | * ** | * ** | * ** |
| Interaction | * ** | | | |

* $P < 0.05$
** $P < 0.01$

TABLE 2

ANTHROPOMETRIC DATA AND RESULTS

OF EXERCISE TESTS

| | Subjects | Age years | Height cm. | Weight kg. | $\dot{M}VO_2$ l/min | Submaximal worktime | Maximal time (secs) |
|---|---|---|---|---|---|---|---|
| Active | 1 | 23 | 177.8 | 68.18 | 3.12 | 70 min. | 160 |
| | 2 | 18 | 185.4 | 70.46 | 3.77 | 55 | 162 |
| | 3 | 18 | 172.7 | 67.27 | 3.29 | 140 | 134 |
| | 4 | 18 | 170.2 | 77.27 | 2.51 | 60 | 143 |
| | 5 | 17 | 177.8 | 73.64 | 4.04 | 60 | 158 |
| | 6 | 19 | 176.5 | 71.36 | 3.09 | 80 | 144 |
| mean | | 19 | 176.5 | 71.36 | 3.31 | 77.5 | 150 |
| Semi-active | 7 | 22 | 174.3 | 68.43 | 2.40 | 50 | 150 |
| | 8 | 23 | 168.9 | 62.31 | 2.31 | 76 | 146 |
| | 9 | 21 | 180.4 | 78.38 | 2.79 | 49 | 153 |
| | 10 | 19 | 173.9 | 74.71 | 2.66 | 18 | 158 |
| | 11 | 20 | 181.3 | 79.88 | 2.84 | 73 | 156 |
| | 12 | 19 | 184.4 | 78.46 | 2.71 | 15 | 155 |
| mean | | 21 | 177.2 | 73.70 | 2.62 | 47 | 153 |
| Sedentary | 13 | 21 | 177.8 | 72.73 | 2.57 | 60 | 140 |
| | 14 | 21 | 181.6 | 80.91 | 2.29 | 40 | 165 |
| | 15 | 21 | 177.8 | 60.46 | 2.29 | 35 | 154 |
| | 16 | 19 | 180.3 | 70.00 | 2.43 | 55 | 143 |
| | 17 | 20 | 188.0 | 70.00 | 2.00 | 60 | 174 |
| | 18 | 24 | 168.9 | 72.27 | 2.71 | 40 | 174 |
| mean | | 21 | 177.8 | 71.36 | 2.38 | 48.3 | 158 |

Effects of Training, Activity and Exercise Upon Lactate Response

Training

The results indicate that the degree of training does not effect the lactate response significantly to alter mean lactate production.

Activity

Examination of the results suggests that there is a significant difference in mean lactate response due to the working or nonworking activity of the muscle. Table 18, Summary Table AB ( Appendix B ) indicates that the working limb alters the lactate response significantly more than the nonworking limb.

Exercise

Table 18, Summary Table AC ( Appendix B ) illustrates the profound effect exercise has on the lactate response. Although the difference between the final and recovery values is not significant, it is evident that the resting data differs significantly from both the final and recovery totals. The first period corresponds to the resting state while the final condition results from short-term exhaustive exercise. The recovery period gives an indication of the rate of removal of lactate from the system.

Interaction

The working limb shows a greater production and removal of plasma lactate than the nonworking limb. This observation indicates that the lactate response changed due to some interaction of these factors. Since there was no other

TABLE 3

MEAN PLASMA LACTATE LEVELS (mg%) DURING MAXIMAL EXERCISE FOR THE WORKING LIMB SAMPLE

| Group | n | Initial | Final | Recovery |
|---|---|---|---|---|
| Trained | 6 | 9.1 ± 1.06 | 60.3 ± 4.08 | 41.0 ± 3.26 |
| Semi-trained | 6 | 7.5 ± .97 | 52.5 ± 7.55 | 52.5 ± 7.63 |
| Sedentary | 6 | 6.0 ± 1.05 | 44.8 ± 11.10 | 41.5 ± 8.01 |

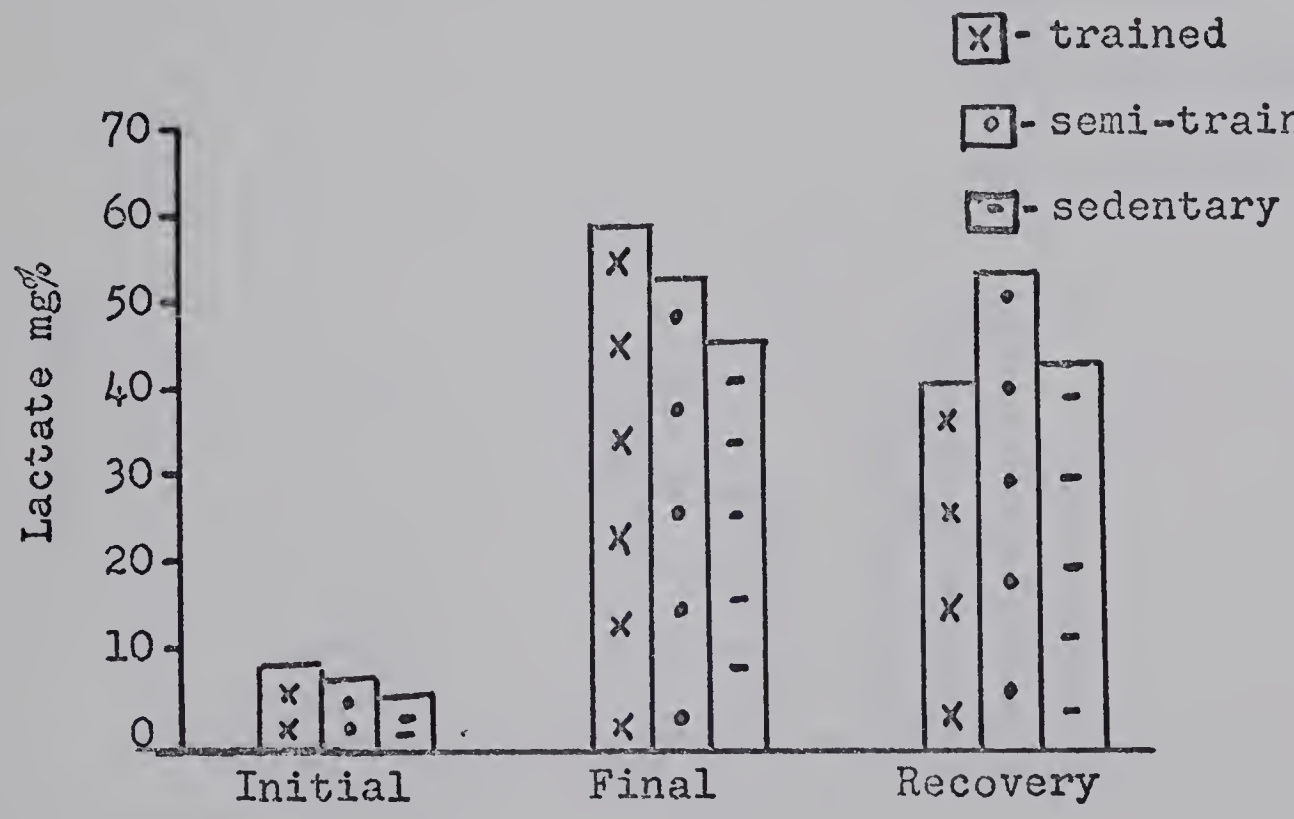



FIGURE 4

MEAN PLASMA LACTATE LEVELS (mg%) DURING MAXIMAL EXERCISE FOR THE WORKING LIMB SAMPLE

TABLE 4

MEAN PLASMA LACTATE LEVELS (mg%) DURING MAXIMAL EXERCISE FOR THE NONWORKING LIMB SAMPLE

| Group | n | Initial | Final | Recovery |
|---|---|---|---|---|
| Trained | 6 | 8.3 ± 1.13 | 36.5 ± 5.44 | 35.5 ± 2.66 |
| Semi-trained | 6 | 8.9 ± 1.50 | 39.3 ± 5.95 | 46.5 ± 7.13 |
| Sedentary | 6 | 6.6 ± 1.29 | 24.4 ± 5.6 | 29.9 ± 2.09 |

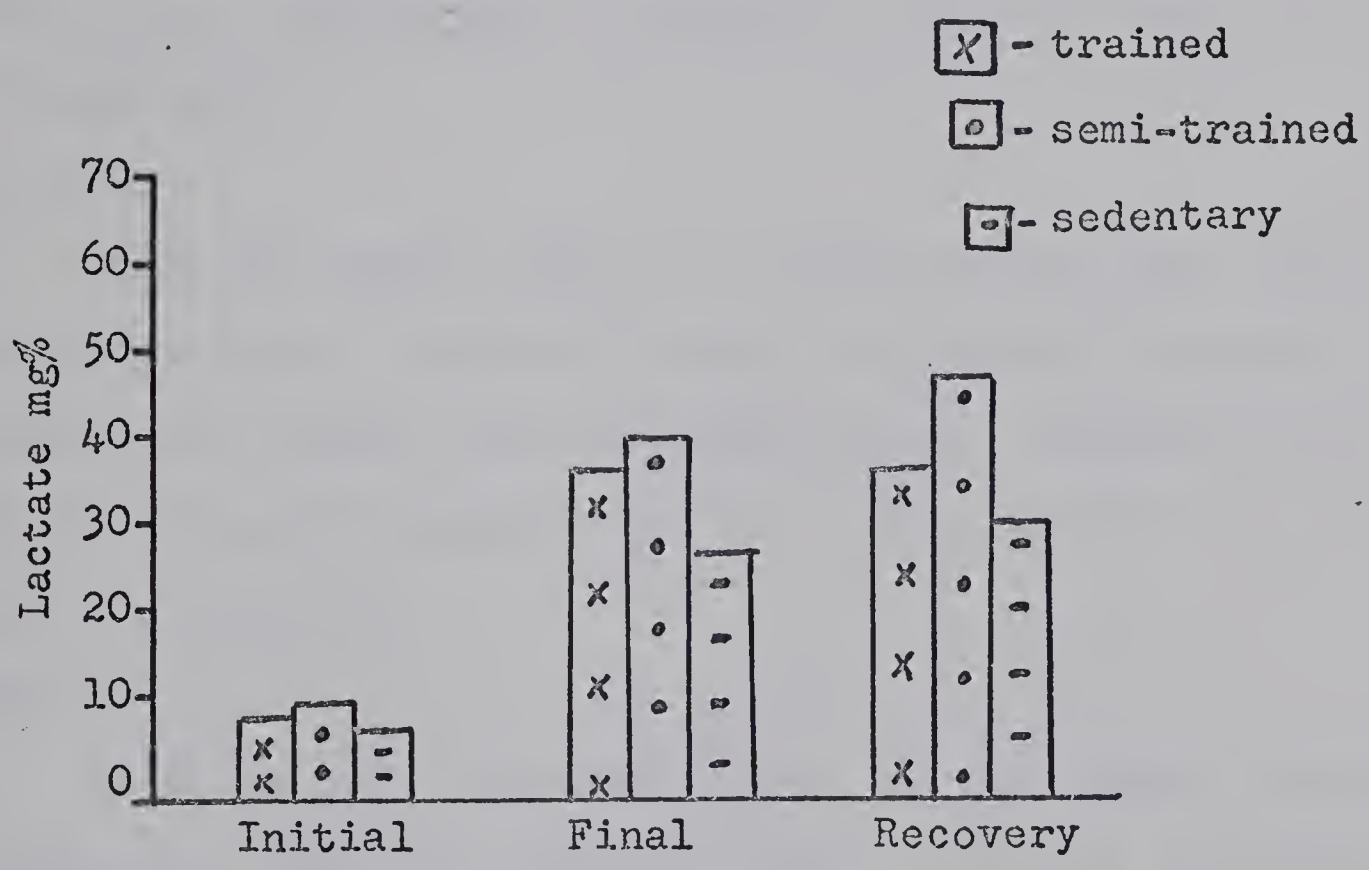



FIGURE 5

MEAN PLASMA LACTATE (mg%) DURING MAXIMAL EXERCISE FOR THE NONWORKING LIMB SAMPLE

significant interaction, this suggests that the trends in lactate response were consistent throughout the trials.

Effect of Training, Activity and Exercise Upon Pyruvate Response

Training

Statistical analysis revealed that a significant difference between the groups occurred in the working limb during the resting state. This implies that the difference in means is due to the training level of the group. All other group comparisons of pyruvate response proved to be insignificant.

Activity

Pyruvate Summary Table 19, Summary Table AB ( Appendix B ) indicates almost identical totals in pyruvate response comparing the working and nonworking limbs. Figures 16, 17, and 18 certainly suggest that the trends within the groups are very similar.

Exercise

Exercise has a profound effect on the plasma pyruvate response as Table 19, Summary Tables AC and BC indicate. The period totals ( Initial, Final and Recovery ) clearly portray this period effect.

Interaction

No interaction indicates that the pyruvate response was similar for the three groups.

TABLE 5

MEAN PLASMA PYRUVATE LEVELS (mg%) DURING MAXIMAL EXERCISE FOR THE WORKING LIMB SAMPLE

| Group | n | Initial | Final | Recovery |
|---|---|---|---|---|
| Trained | 6 | .84 ± .08 [a] | 1.33 ± .11 | 2.65 ± .07 |
| Semi-trained | 6 | .65 ± .08 | 1.20 ± .21 | 2.55 ± .09 |
| Sedentary | 6 | .39 ± .04 [w] | 1.57 ± .30 | 2.29 ± .23 |

[a] indicates significant differences between groups ($P<0.05$)

[w] Tukey's w revealed the sedentary group to be significantly different from the trained and semi-trained groups

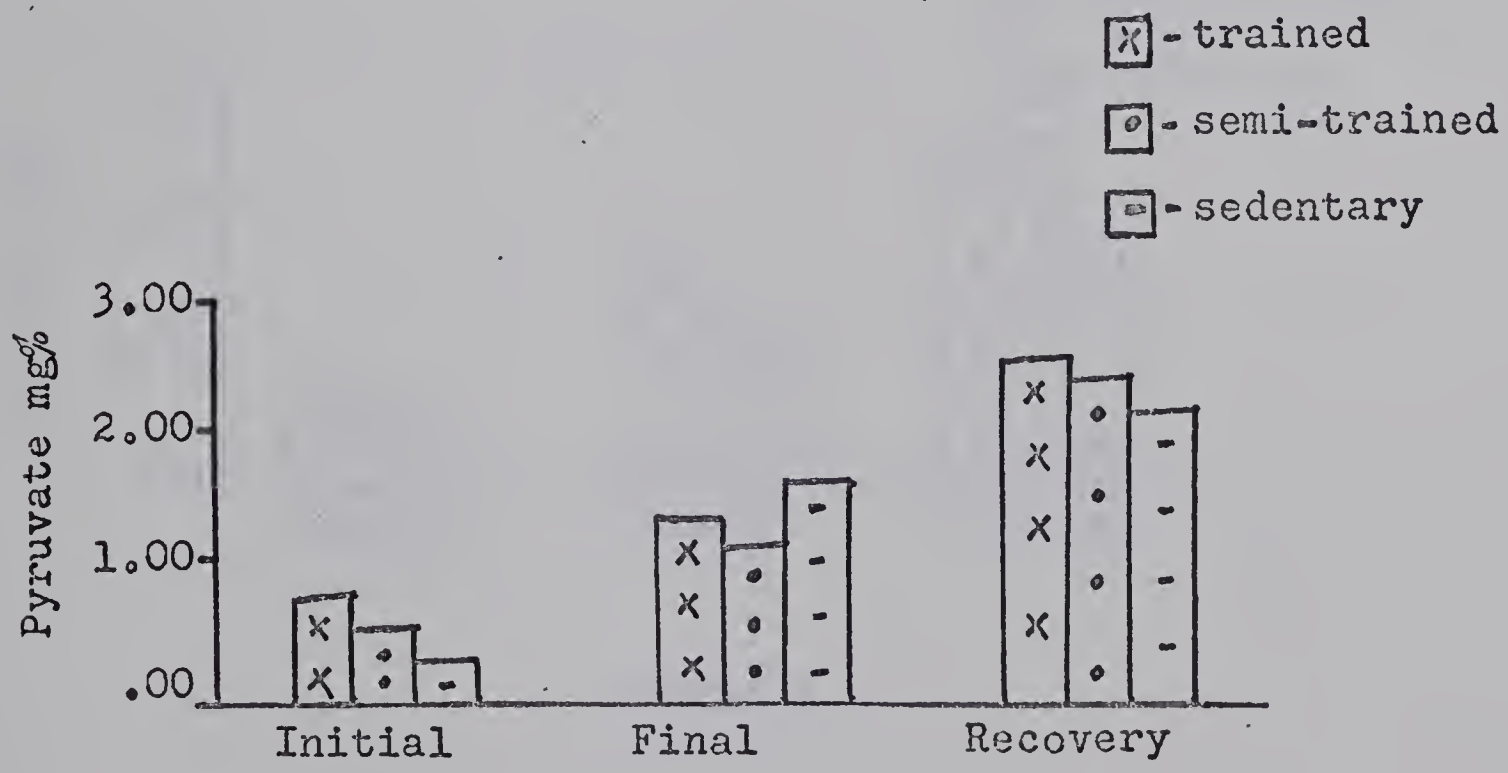



FIGURE 6

MEAN PLASMA PYRUVATE (mg%) DURING MAXIMAL EXERCISE FOR THE WORKING LIMB SAMPLE

TABLE 6

MEAN PLASMA PYRUVATE LEVELS (mg%) DURING MAXIMAL EXERCISE FOR THE NONWORKING LIMB SAMPLE

| Group | n | Initial | Final | Recovery |
|---|---|---|---|---|
| Trained | 6 | .75 ± .08 | 1.63 ± .25 | 2.50 ± .28 |
| Semi-trained | 6 | .65 ± .09 | 1.03 ± .15 | 2.54 ± .09 |
| Sedentary | 6 | .53 ± .10 | 1.19 ± .20 | 1.84 ± .19 |

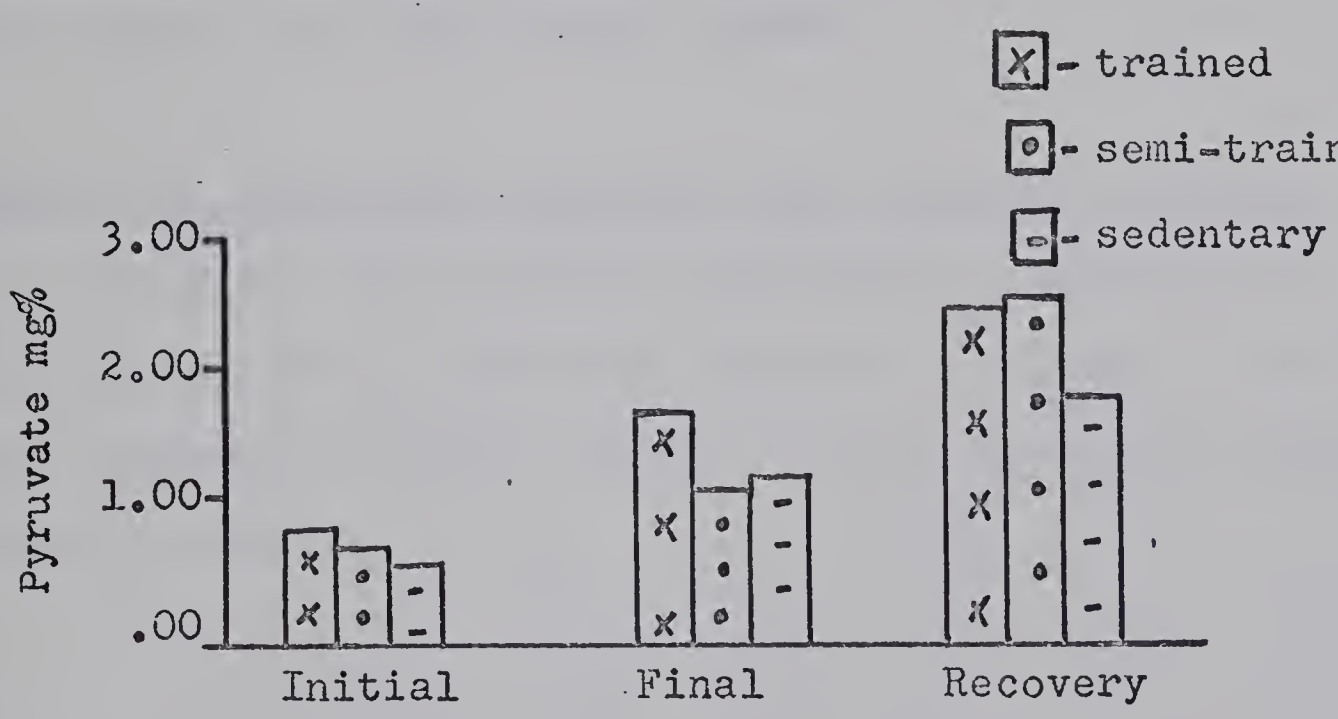



FIGURE 7

MEAN PLASMA PYRUVATE (mg%) DURING MAXIMAL EXERCISE FOR THE NONWORKING LIMB SAMPLE

Effects of Submaximal Exercise Upon Lactate Response

Figures 8 and 9 which portray the group responses for the working and nonworking limbs respectively, indicate appreciable lactate activity in all groups throughout the submaximal sessions. The sedentary group is notably less responsive in lactate concentration changes than either of the trained or semi-trained groups. The latter two groups appear to respond similarly to a submaximal stress. It is interesting to note that in all instances just prior to exhaustion, there is a considerable increase in lactate concentration. This response is particularly evident in the trained and semi-trained groups.

Effects of Submaximal Exercise Upon Pyruvate Response

Pyruvate concentration progressively increases in all groups during submaximal exercise ( Figures 10 and 11 ). The response is notably greater in the trained and semi-trained groups.

TABLE 7

MEAN PLASMA LACTATE LEVELS (mg%) DURING SUBMAXIMAL EXERCISE FOR THE WORKING LIMB SAMPLE

| Group | n | Initial | 25% | 50% | 75% | Final | Recovery |
|---|---|---|---|---|---|---|---|
| Trained | 6 | 7.6 | 19.3* | 17.3 | 27.5 | 40.3[a] | 18.9 |
| Semi-trained | 6 | 6.9 | 13.6 | 19.9 | 27.1 | 40.6 | 29.2 |
| Sedentary | 6 | 5.5 | 14.0 | 18.3 | 19.1 | 23.7 | 14.0 |

[a] indicates significant difference between the groups

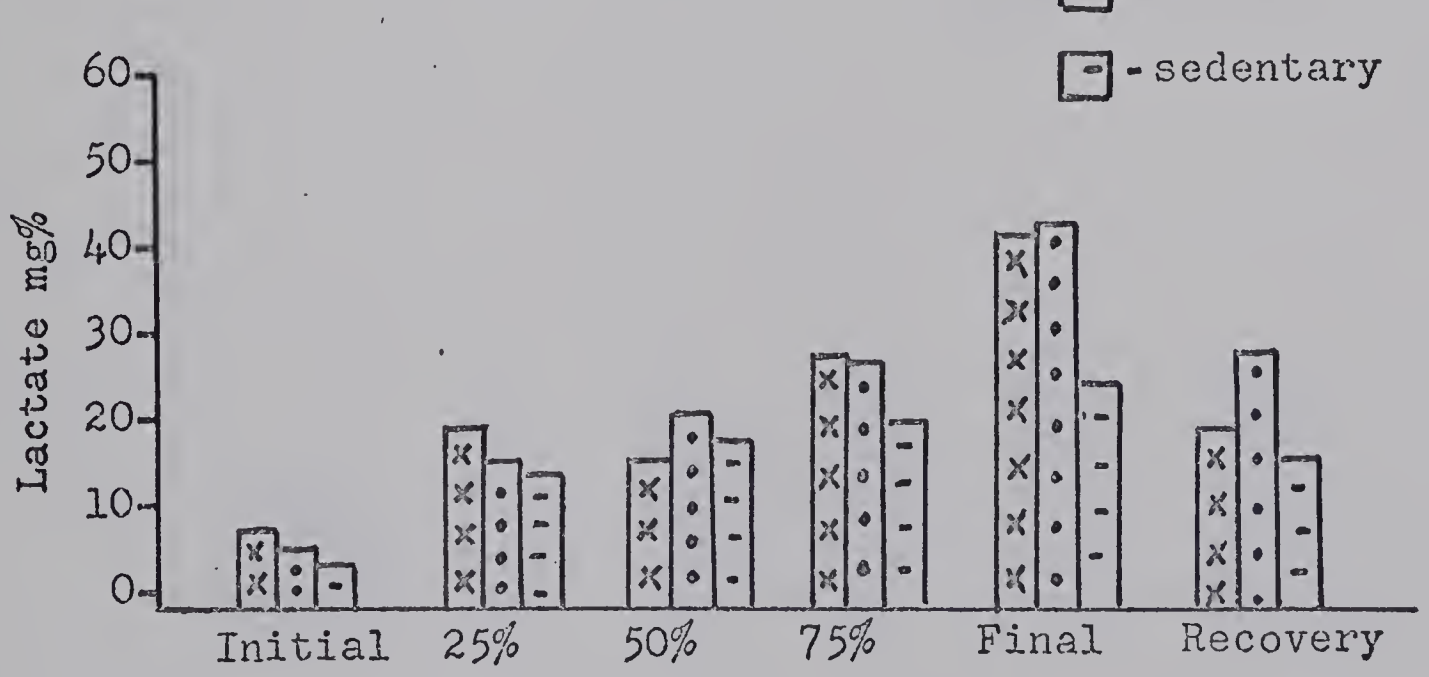



FIGURE 8

MEAN PLASMA LACTATE (mg%) DURING SUBMAXIMAL EXERCISE FOR THE WORKING LIMB SAMPLE

* means derived by linear interpolation with reference to percentage of exercise time.

TABLE 8

MEAN PLASMA LACTATE LEVELS (mg%) DURING SUBMAXIMAL EXERCISE FOR THE NONWORKING LIMB SAMPLE

| Group | n | Initial | 25% | 50% | 75% | Final | Recovery |
|---|---|---|---|---|---|---|---|
| Trained | 6 | 10.7 | 19.5* | 17.3 | 22.5 | 37.1[a] | 22.3 |
| Semi-trained | 6 | 7.2 | 12.2 | 17.0 | 21.6 | 31.0 | 20.2 |
| Sedentary | 6 | 5.6 | 11.1 | 17.1 | 16.6 | 22.1 | 20.6 |

[a] indicates significant difference between the groups

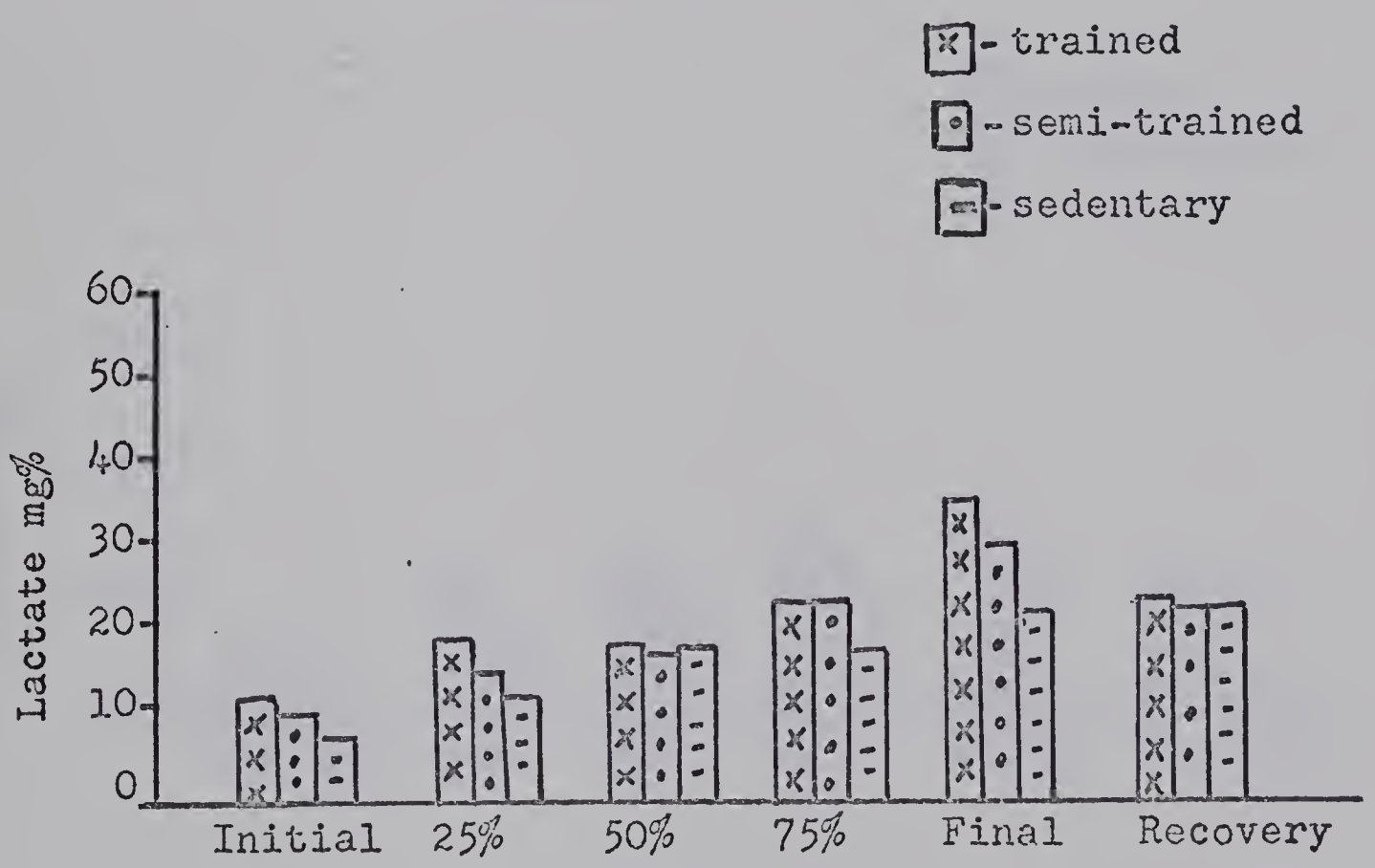



FIGURE 9

MEAN PLASMA LACTATE (mg%) DURING SUBMAXIMAL EXERCISE FOR THE NONWORKING LIMB

* means derived by linear interpolation with reference to percentage of exercise time.

TABLE 9

MEAN PLASMA PYRUVATE LEVELS (mg%) DURING SUBMAXIMAL EXERCISE FOR THE WORKING LIMB SAMPLE

| Group | n | Initial | 25% | 50% | 75% | Final | Recovery |
|---|---|---|---|---|---|---|---|
| Trained | 6 | .52 | 1.09 *a | 1.27 | 1.47 | 1.80 | 1.18 |
| Semi-trained | 6 | .38 | .69 | 1.04 | 1.30 | 1.41 | 1.17 |
| Sedentary | 6 | .49 | .69 | .88 | 1.01 | 1.20 | .89 |

a indicates significant difference between the groups

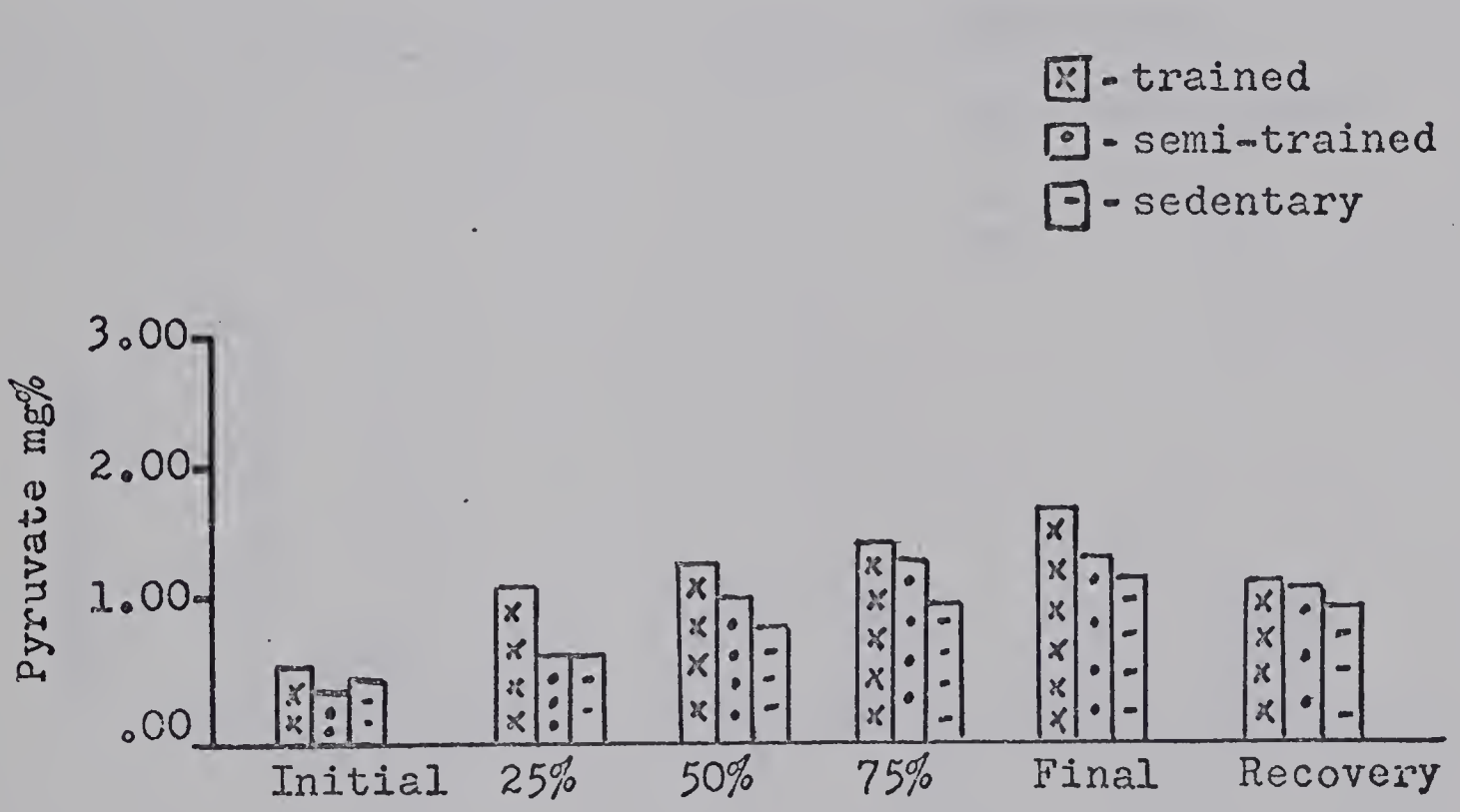



FIGURE 10

MEAN PLASMA PYRUVATE (mg%) DURING SUBMAXIMAL EXERCISE FOR THE WORKING LIMB SAMPLE

* means derived by linear interpolation with reference to percentage of exercise time.

TABLE 10

MEAN PLASMA PYRUVATE LEVELS (mg%) DURING SUBMAXIMAL EXERCISE FOR THE NONWORKING LIMB SAMPLE

| Group | n | Initial | 25% | 50% | 75% | Final | Recovery |
|---|---|---|---|---|---|---|---|
| Trained | 6 | .54 | .86* | 1.00 | 1.08 | 1.47 | 1.15 |
| Semi-trained | 6 | .47 | .75 | .85 | 1.08 | 1.09 | 1.02 |
| Sedentary | 6 | .49 | .72 | .85 | .89 | 1.23 | 1.10 |

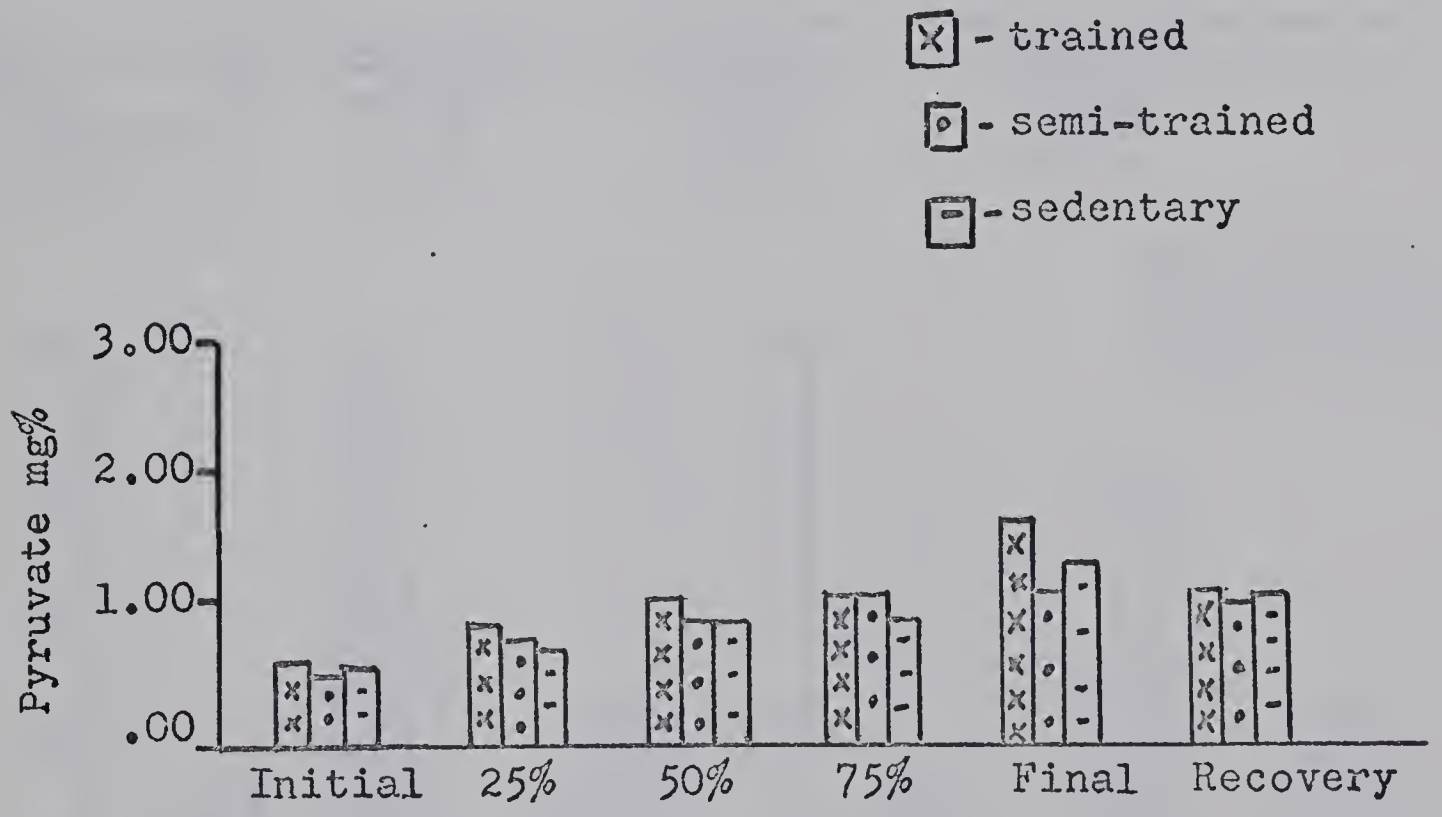



FIGURE 11

MEAN PLASMA PYRUVATE (mg%) DURING SUBMAXIMAL EXERCISE FOR THE NONWORKING LIMB SAMPLE

* means derived by linear interpolation with reference to percentage of exercise time.

TABLE 11

MEAN LACTATE/PYRUVATE RATIOS

Working Sample

| Group: | n | Initial | Final | Recovery |
|---|---|---|---|---|
| Trained | 6 | 11.25 | 45.37 | 14.98 |
| Semi-trained | 6 | 11.76 | 43.80 | 20.49 |
| Sedentary | 6 | 15.92 | 26.57 | 15.39 |

Nonworking Sample

| Group: | n | Initial | Final | Recovery |
|---|---|---|---|---|
| Trained | 6 | 11.70 | 22.42 | 16.20 |
| Semi-trained | 6 | 13.70 | 38.10 | 18.28 |
| Sedentary | 6 | 12.47 | 20.63 | 16.49 |

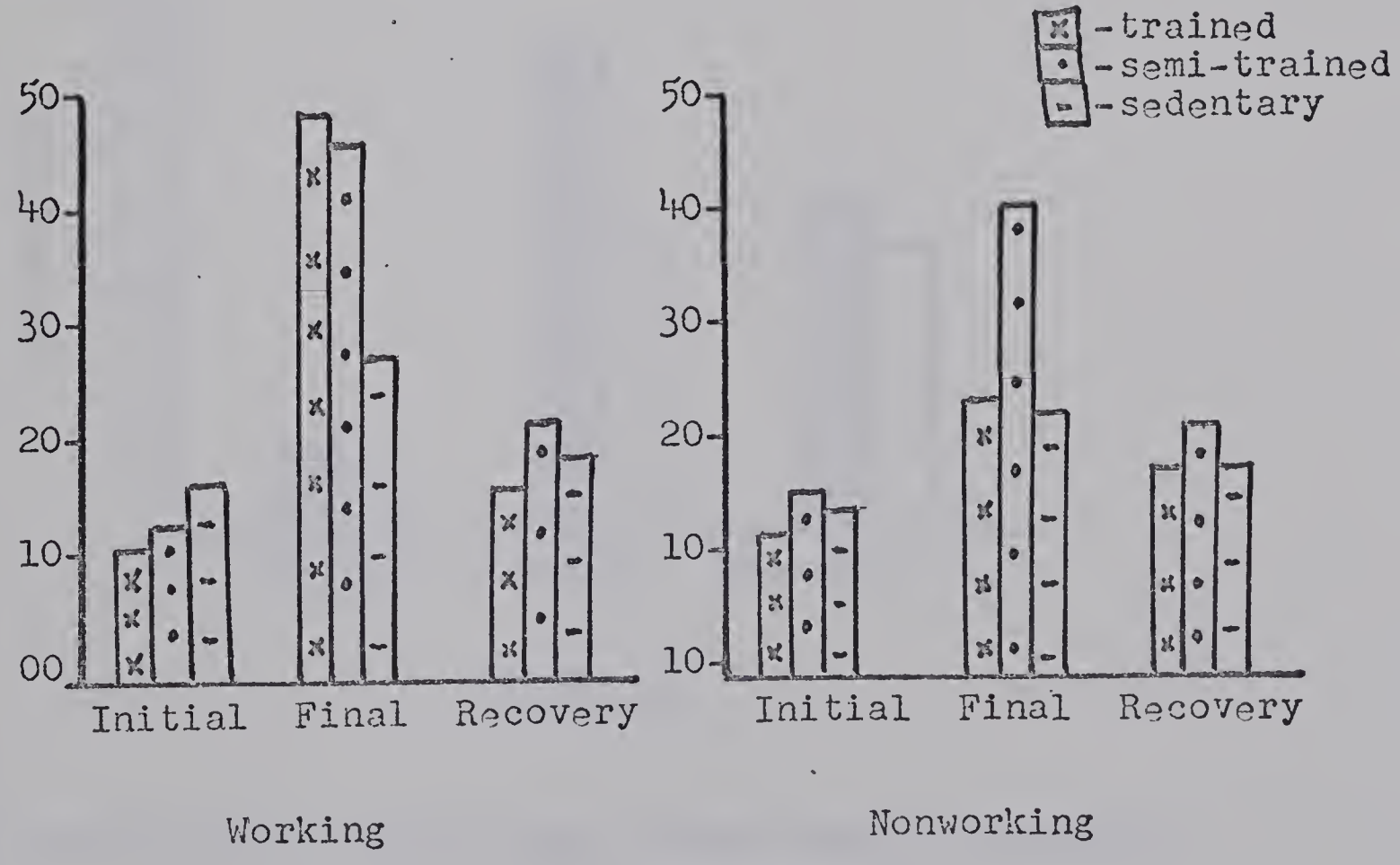



FIGURE 12

MEAN LACTATE/PYRUVATE RATIOS FOR WORKING AND NONWORKING LIMBS

TABLE 12

MEAN PLASMA LACTATE LEVELS (mg%) DURING MAXIMAL EXERCISE FOR TRAINED INDIVIDUALS

| Sample | n | Initial | Final | Recovery |
|---|---|---|---|---|
| Working | 6 | 9.1 ± 1.06 | 60.3 ± 4.03 [a] | 41.0 ± 3.26 |
| Nonworking | 6 | 8.3 ± 1.13 | 36.5 ± 5.44 | 35.5 ± 2.66 |

[a] indicates significant difference between working and nonworking limbs

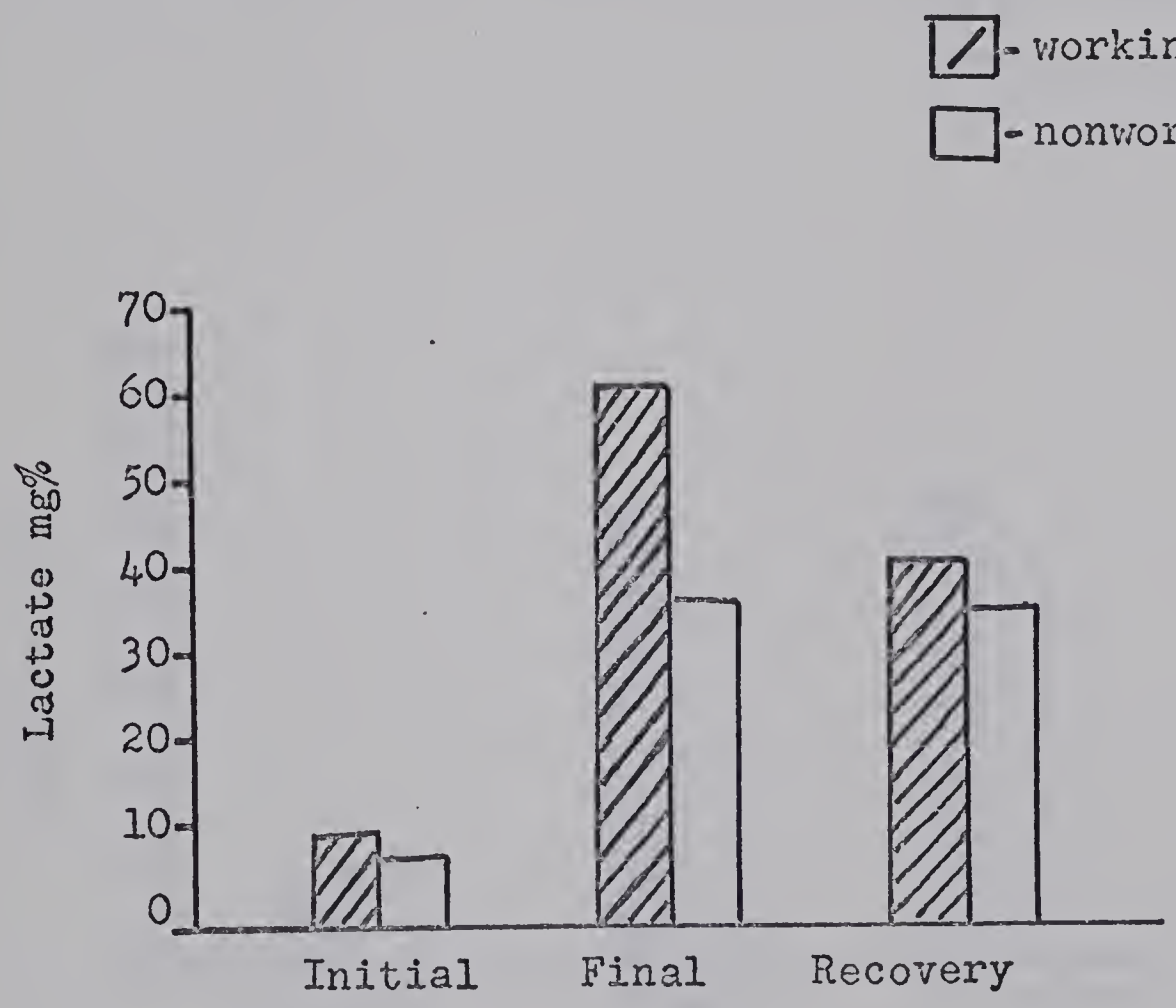



FIGURE 13

MEAN PLASMA LACTATE (mg%) DURING MAXIMAL EXERCISE FOR TRAINED INDIVIDUALS

TABLE 13

MEAN PLASMA LACTATE LEVELS (mg%) DURING MAXIMAL EXERCISE FOR SEMI-TRAINED INDIVIDUALS

| Sample | n | Initial | Final | Recovery |
|---|---|---|---|---|
| Working | 6 | 7.5 ± .97 | 52.5 ± 7.55 | 52.5 ± 7.63 |
| Nonworking | 6 | 8.9 ± 1.50 | 39.3 ± 5.95 | 46.5 ± 7.13 |

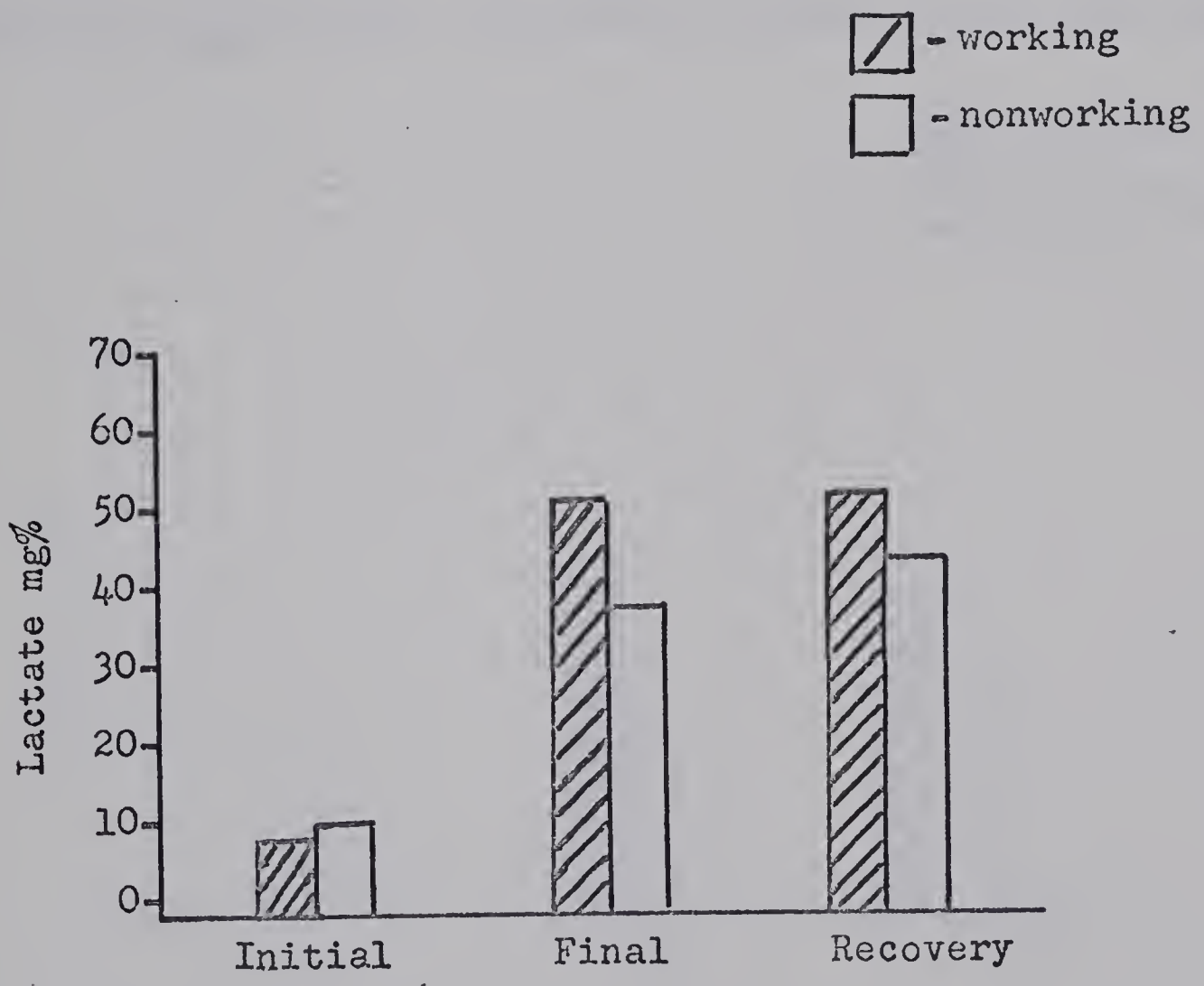



FIGURE 14

MEAN PLASMA LACTATE (mg%) DURING MAXIMAL EXERCISE FOR SEMI-TRAINED INDIVIDUALS

TABLE 14

MEAN PLASMA LACTATE LEVELS (mg%) DURING MAXIMAL EXERCISE FOR SEDENTARY INDIVIDUALS

| Sample | n | Initial | Final | Recovery |
|---|---|---|---|---|
| Working | 6 | 6.05 ± 1.15 | 44.8 ± 11.1[a] | 41.5 ± 8.01 |
| Nonworking | 6 | 6.60 ± 1.29 | 24.4 ± 5.6 | 29.9 ± 2.08 |

[a] indicates significant difference between working and nonworking limbs

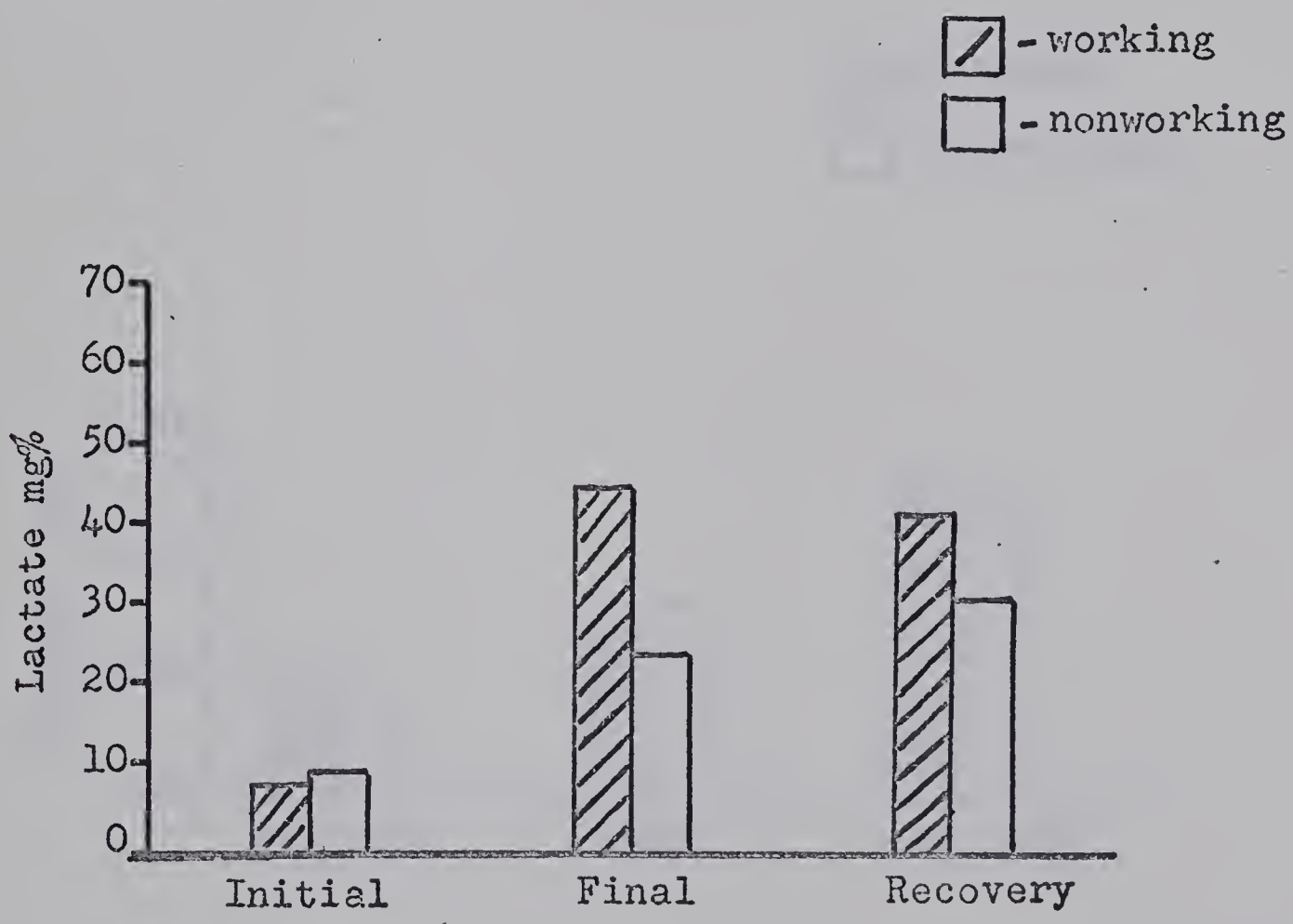



FIGURE 15

MEAN PLASMA LACTATE (mg%) DURING MAXIMAL EXERCISE FOR SEDENTARY INDIVIDUALS

TABLE 15

MEAN PLASMA PYRUVATE LEVELS (mg%) DURING MAXIMAL EXERCISE FOR TRAINED INDIVIDUALS

| Sample | n | Initial | Final | Recovery |
|---|---|---|---|---|
| Working | 6 | .84 ± .08 | 1.33 ± .11 | 2.65 ± .07 |
| Nonworking | 6 | .75 ± .08 | 1.63 ± .25 | 2.50 ± .16 |

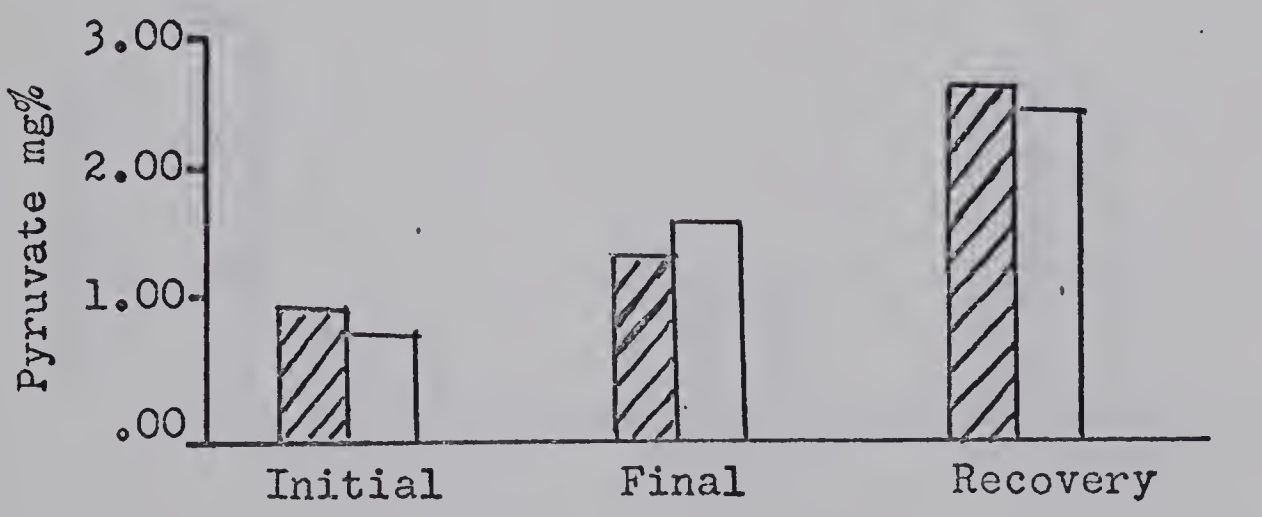



FIGURE 16

MEAN PLASMA PYRUVATE (mg%) DURING MAXIMAL EXERCISE FOR TRAINED INDIVIDUALS

TABLE 16

MEAN PLASMA PYRUVATE LEVELS (mg%) DURING MAXIMAL EXERCISE FOR SEMI-TRAINED INDIVIDUALS

| Sample | n | Initial | Final | Recovery |
|---|---|---|---|---|
| Working | 6 | .65 ± .09 | 1.20 ± .21 | 2.55 ± .09 |
| Nonworking | 6 | .65 ± .08 | 1.03 ± .15 | 2.54 ± .09 |

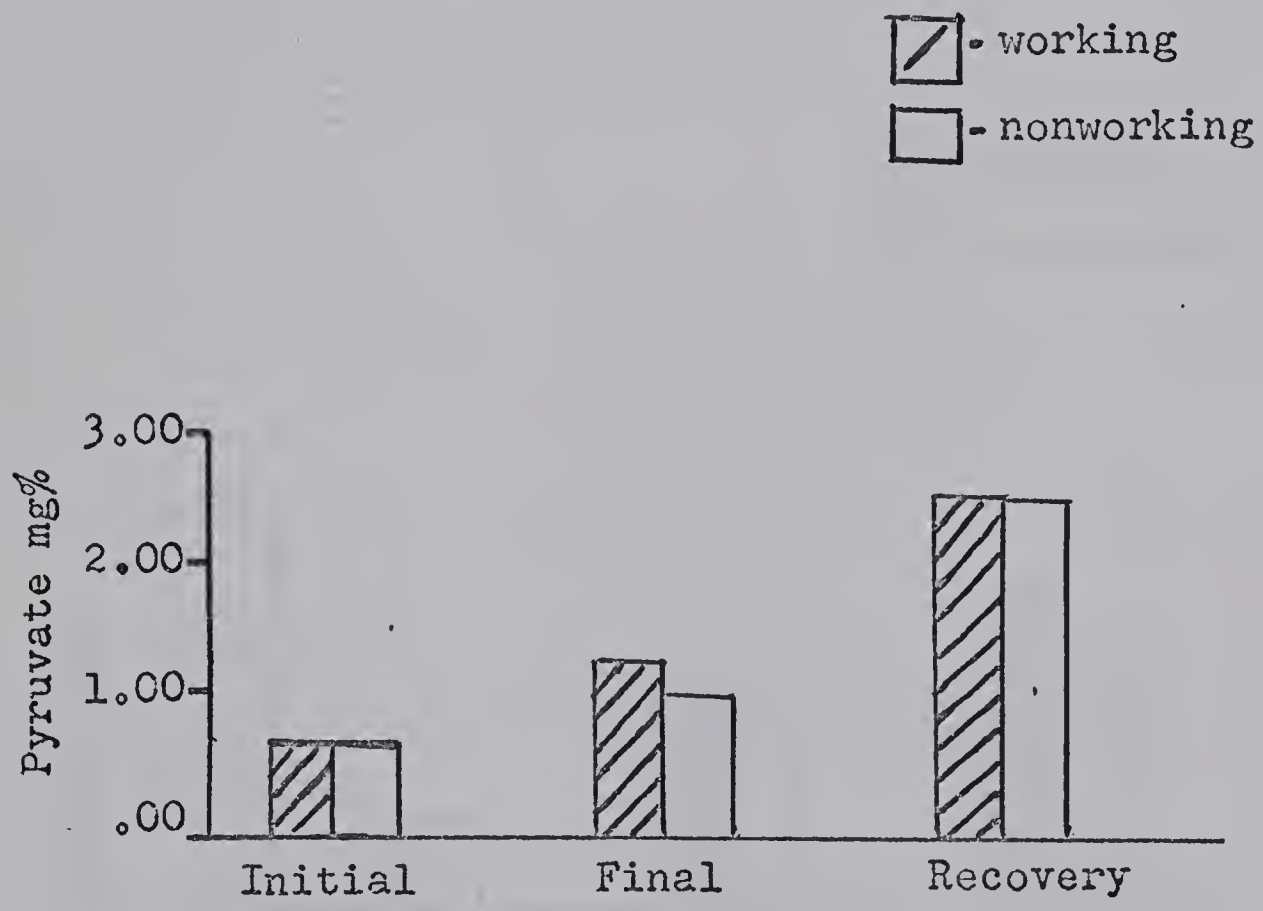



FIGURE 17

MEAN PLASMA PYRUVATE (mg%) DURING MAXIMAL EXERCISE FOR SEMI-TRAINED INDIVIDUALS

TABLE 17

MEAN PLASMA PYRUVATE LEVELS (mg%) DURING MAXIMAL EXERCISE FOR SEDENTARY INDIVIDUALS

| Sample | n | Initial | Final | Recovery |
|---|---|---|---|---|
| Working | 6 | .39 ± .04 | 1.57 ± .30 | 2.29 ± .23 |
| Nonworking | 6 | .53 ± .10 | 1.19 ± .20 | 1.84 ± .19 |

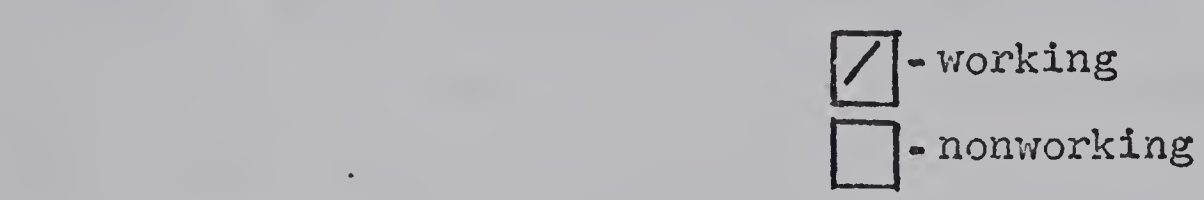



FIGURE 18

MEAN PLASMA PYRUVATE (mg%) DURING MAXIMAL EXERCISE FOR SEDENTARY INDIVIDUALS

# CHAPTER V

## DISCUSSION

The few significant results obtained do not allow generalizations to be made to similar populations since disagreement with other reports is evident. The effects of acute maximal exercise on the plasma pyruvate and lactate concentrations are well known. In this study, no evidence was found to suggest that the oxygen debt was being repaid during the exercise. These results are in agreement with Rowell *et al* ( 68 ), Knuttgen ( 49 ), and Wasserman *et al* ( 78 ), that venous lactate concentrations increase with work time. Therefore, discussions will be limited to the effects of training, limb activity ( working and nonworking ), and submaximal exercise on the lactate and pyruvate response.

### The Effect of Training and Submaximal Exercise on the Lactate and Pyruvate Response

Margaria *et al* ( 56 ), Barnard ( 9 ), as well as Williams ( 81 ) found a decrease in lactate response due to training. They attribute this decrease in lactate production to the greater aerobic capacities of the athlete. Alterations in enzyme activity ( 14,38,39,40 ) and mitochondrial density ( 32 ) certainly suggest possibilities for accelerated pyruvate oxidations. With pyruvate removal being more efficient as a result of training, one would expect lactate

production to be curtailed. The evidence here, however, indicates that there was no significant difference between the groups ( $P<0.01$ ) with respect to lactate response. In fact, the trained group consistently responded with greater lactate production than either of the other groups. This response was evident during both maximal and submaximal sessions. One can argue, that the trained group had higher workloads than the other groups and therefore, lactate production should be greater. However, workloads were deliberately selected with consideration for the subjects $\dot{M}VO_2$.

If training ( as reflected by mitochondrial density and enzymic adaptations ) influences lactate concentration, one would expect the trained group to respond with a reduction in metabolite values during submaximal exercise. This was not found. There was a progressive increase in both lactate and pyruvate production during the submaximal sessions. These results contradict the findings of Saiki *et al* ( 70 ) who suggest that the alactacid oxygen debt is repaid during steady state conditions. Astrand *et al* ( 7 ) found a successive decrease in blood lactate concentrations with work time despite a maximal effort by the cross country skiers. We found an obvious increase in lactate and pyruvate production in all groups that was particularly evident prior to exhaustion. Cunninghams finding ( 17 ), that lactate concentrations increase as a result of training support these results.

One can suggest, that the training program was not

adequate or long enough to produce the effective enzymatic or mitochondrial adaptations expected. However, this still does not explain why the trained group's mean performance time ( $\overline{X}$ = 77.5 min. ) was significantly longer than either the semi-trained ( $\overline{X}$ = 47 min. ) or sedentary ( $\overline{X}$ = 48.3 min. ) groups submaximal performance times. The physiological explanation is obscure.

The Effect of Limb Activity (working vs nonworking ) on The Lactate and Pyruvate Response

The plasma lactate response for the working limb during maximal exercise was significantly different from the non-working limb in the trained and sedentary groups. This response is to be expected since muscular activity creates a hypoxic condition conducive to lactate production. Why the response was not evident in the semi-trained group is unclear. A rapid equilibrium of the metabolites was attained, however, that was similar for all groups. The working limb of the trained group is notably more responsive during recovery than the working limb for either of the other groups. This response to take up lactate at a rate appreciably greater than the semi-trained or sedentary groups suggests that local adaptations have taken place in the trained group. Indeed, the training program must have been intense enough to bring about expected physiological changes.

Plasma lactate and pyruvate responses in working and

nonworking limbs during submaximal exercise are almost identical. These results do not agree with Rowell et al ( 68 ) or Keul ( 47 ) who found nonworking values to be higher than working values. Certainly, the lactate and pyruvate responses in this instance are not reflecting local hypoxic conditions. This lack of response to local tissue hypoxia suggest that during exercise, the blood may function as an independent tissue regulating its own concentrations of lactate and pyruvate. Perhaps excessive changes in these metabolites become noticeable during recovery when the hypoxic state of the tissues change abruptly.

The trained group lasted considerably longer than the other groups while exercising submaximally ( $\bar{X}$ = 77.5 min. compared to $\bar{X}$ = 47 min. and $\bar{X}$ = 48.3 min. ) despite considerable lactate and pyruvate response. If mitochondrial and/or enzymatic adaptations result from training, a decrease in lactate and pyruvate response should be expected. Precisely because workloads were related to the subjects $\dot{M}VO_2$, lactate and pyruvate production should reflect this capacity. The individual with the greater aerobic capacity should keep lactate production curtailed. The results here indicate exactly the opposite of what was expected. The trained and semi-trained groups display considerable metabolite production while the sedentary group is less responsive to concentration changes of lactate and pyruvate.

To account for the longer submaximal exercise time

in the trained group, despite considerable lactate and pyruvate production, an analysis of the blood itself might provide a partial explanation. Pyruvate concentrations are consistently greater in the trained group throughout the submaximal trials. Availability of pyruvate in the red blood cell as a proton acceptor forming lactate reflects increased concentrations of $NAD^+$. $NAD^+$ is essential for the production of 2,3-DPG, which is known to facilitate oxygen release at the tissue level. In addition, deoxy-hemoglobin ( abundant during exercise ) promotes production of 2,3-DPG. The semi-trained and sedentary groups with lower concentrations of pyruvate available to form lactate would have corresponding lower concentrations of red blood cell $NAD^+$. This lower concentration of pyruvate suggests that there would not be as much $NAD^+$ available for production of 2,3-DPG. A decrease in 2,3-DPG in the red blood cells of the sedentary and semi-trained groups might conceivably reduce oxygen release. This lack of 2,3-DPG ( a reflection of pyruvate concentration ) may be one of the contributing factors to the shorter performance times of the sedentary and semi-trained groups.

# CHAPTER VI

## SUMMARY AND CONCLUSION

### Summary

Eighteen male University of Alberta students were investigated to determine the effect of training on the concentrations of plasma lactate and pyruvate. Three groups were compared under maximal and submaximal conditions. These were highly trained, sem-trained and sedentary groups. Arterial and venous blood samples were taken at rest, during the exercise session, at the point of exhaustion and during the recovery period. The samples were analyzed for plasma concentrations of lactate and pyruvate. Comparisons were made within the groups for possible differences in arterial and venous samples.

The results indicate ( $P<0.01$ ) that there was no difference in group response during exercise with respect to alterations in plasma lactate concentrations. During the recovery period, however, the trained group responded with accelerated lactate uptake in the working limb indicating that training resulted in local tissue adaptations. The pyruvate response proved to differ significantly in the three groups. This difference, however, was evident only during the resting state.

During the submaximal trials, the plasma concentrations of lactate and pyruvate in working and nonworking limbs were almost identical.

Conclusion

With respect to maximal and submaximal exercise, the following conclusions were drawn.

( 1 ). The 8 week training program was not sufficient to produce significant group differences in the concentration of plasma lactate.

( 2 ). A significant difference between the groups in the concentration of plasma pyruvate was evident during the resting state and throughout the submaximal session. This suggests that training resulted in an adaptive response possibly within the muscle or the blood itself.

# REFERENCES


1. Alpert, Norman, R., " Lactate production and removal and the regulation of metabolism ", Annals of the New York Academy of Sciences, 119: 995-1012, 1965.

2. Andres, R., Cader, G., Zierler, K., " The quantitatively minor role of carbohydrates in oxidative metabolism by skeletal muscle in intact man in the basal state. Measurements of oxygen and glucose uptake and carbon dioxide and lactate production in the forearm ", Journal of Clinical Investigation, 35: 671-683, 1956.

3. Astrand, Irma, " Aerobic work capacity in men and women with special reference to age ", Acta Physiologica Scandinavica, 49: 45-60, 1960.

4. Astrand, Irma, Astrand, P.O., and K. Radahl, " Maximal heart rate during work in older men ", Journal of Applied Physiology, 14: 562-566, 1959.

5. Astrand, P.O., Experimental Studies in Physical Working Capacity in Relation to Sex and Age, Copenhagen: Munksgaard, 1952.

6. Astrand, P.O., " Intermittent muscular work ", Acta Physiologica Scandinavica, 48: 448-453, 1960.

7. Astrand, P.O., I. Hallback, R. Hedman and B. Saltin, " Blood lactates after prolonged severe exercise " Journal of Applied Physiology, 18: 617-622, 1963.

8. Barnard, James R., Merle L. Foss, and Charles M. Tipton, " Oxygen debt: involvement of the Cori Cycle " , Int. Z. angew. Physiol, 28: 105-19, 1970.

9. Barnard, James R., and Kenneth M. Baldwin, " The effect of training and various workloads on the lactacid-alactacid oxygen debt response of exercising dogs ", Int. Z. angew Physiol, 28: 120-30, 1970.

10. Barr, David P., Himwich, H.E., Green, R.P., " Studies in the physiology of muscular exercise ", Journal of Biological Chemistry, 55: 495-515, 1923.

11. Bergstrom, Jonas, " Muscle glycogen syntesis after exercise: An enhancing factor localized to the muscle cells in man " , Nature, 210: 309-10, 1966.

12. Bergstrom, Jonas, Hultman, E., " A study of the glycogen metabolism during exercise in man ", Scandinavian Journal of Clinical Investigation, 19: 218-28, 1967.

13. Bock, A.V., C. Vancaulaert, D.B. Dill, A. Folling, and L.M. Hurxthal, " Studies in muscular activity-III, Dynamical changes in man at work ", Journal of Physiology, 66: 136-61, 1928.

14. Booth, Michael, Anthony, " Muscle glycogen phosphorylase and training ", Unpublished Master's Thesis, University of Alberta, Edmonton, 1970.

15. Burridge, W.A., " An inquiry into some chemical factors of fatigue ", Journal of Physiology, XLI: 285-307, 1910-11.

16. Burridge, W.A., " Lactic acid and cardiac muscle ", Journal of Physiology, XLIII, xli, 1910.

17. Cunningham, David, A., and John A. Faulkner, " The effect of training on aerobic and anaerobic metabolism during a short exhaustive run ", Medicine and Science in Sports, 1-#2: 65-69, 1969.

18. Cook, L.C., Hurst, R.H., " Blood lactic acid in men during rest ", Journal of Physiology, 79: 443-54, 1933.

19. Depocas , Florent, Yves Minaire, and Josdph Chatonnet, " Rates of formation and oxidation of lactic acid in dogs at rest and during moderate exercise ", Canadian Journal of Physiology and Pharmocology, 47: 603-10, 1969.

20. Diamant, Bertil, Jan Karlsson and Bengt Saltin, " Muscle tissue lactate after exercise in man ", Acta Physiologica Scandinavica, 72: 383-84, 1968.

21. Dill, D.B., Talbot, J.H., Edwards, H.T., " Studies in muscular activity ", Journal of Physiology, 69: 267-305, 1930.

22. DI Prampero, P.E., P. Cerretelli, and J. Piiper, " Lactic acid formation in gastrocnemius muscle of the dog and its relation to oxygen debt contraction ", Respiration Physiology, 8: 347-53, 1970.

23. Doll, E., J. Keul, and C. Maiwald, " Oxygen tension and acid-base equilibrium in venous blood of working muscle ", American Journal of Physiology, 215: #1-23-29, 1968.

24. Drury, D.R., " Chemistry and metabolism of L+ and D- lactic acids ", Annals of the New York Academy of Sciences, 119: art. 3, 1061-69, 1965.

25. Eggleton, Grace, M., C.L. Evans, " The lactic acid content of the blood after muscular contraction under experimental conditions ", Journal of Physiology, 70: 269-93, 1930.

26. Eggleton, Grace M. and Evans, C.L. " The lactic acid formation and removal with change of blood reaction ", Journal of Physiology, 70: 260-68, 1930.

27. Feldman, I., and Hill, L., " The influence of oxygen inhalation on the lactic acid produced during hard work ", Journal of Physiology, 42: 439-43, 1911.

28. Fletcher, W.M., Hopkins, F.G., " Lactic acid in amphibian muscle ", Journal of Physiology, 35: 247-309, 1907.

29. Fletcher, W.M., " Acid formation in muscle ", Journal of Physiology, 47: 361-80, 1914.

30. Fordtran, J.S., Saltin, Bengt, " Gastric emptying and intestinal absorption during severe prolonged exercise ", Journal of Applied Physiology, 23: #3, 331-35, 1967.

31. Gollnick, Phillip D., " Energy production and lactic acid formation ", Physiological Aspects of Sports and Physical Fitness, American College of Sports Medicine and the Athletic Institute, 16-19, 1968.

32. Gollnick, Phillip D., and King, D.W., " The effect of exercise and training on mitochondrial adaptations ", American Journal of Physiology, 216: 1502, 1969.

33. Guyton, Arthur C., Textbook of Medical Physiology, 3rd ed., W.B. Saunders Company, Philadelphia and London, 1970.

34. Hansen, J.E., G.P. Stelter and J.A. Vogel, " Arterial pyruvate, lactate, pH, and $PCO_2$, during work at sea level and high altitude ", Journal of Applied Physiology, 23: 523-30, 1967.

35. Hartree, W., Hill, A.V., " The recovery heat production in muscle ", Journal of Physiology, 56: 367. 1922.

36. Hewlett, A.W., Barnett, D.G., " The effect of breathing oxygen enriched air during exercise upon pulmonary ventilation and upon the lactic acid content of blood and urine ", Journal of Clinical Investigation, 3: 317-27, 1926-27.

37. Hollock, Phillip, " Lactic acid during rest and after exercise in subjects with various types of heart disease with special references to congenital heart disease ", Journal of Clinical Investigation, 18: 385-94, 1939.

38. Holloszy, J.O., L.B. Oscai, I.J. Don and P.A. Mole, " Mitochondrial citric acid cycle and related enzymes: adaptive response to exercise ", Biochemical and Biophysical Research Communications, 40(6): 1368, 1970.

39. Holloszy, J.O. and Lawerence B. Oscai, " Effect of exercise on α-glycerophosphate dehydrogenase activity in skeletal muscle ", Archives of Biochemistry and Biophysics, 130: 653-56, 1969.

40. Holloszy, J.O., " Biochemical adaptations in muscle: Effects of exercise on mitochondrial oxygen uptake and respiratory enzyme activity in skeletal muscle ", The Journal of Biological Chemistry, 242: 2278-82, 1967.

41. Hill, A,V., " The heat production of surviving amphibian muscle during rest, activity, and rigor ", Journal of Physiology, 44: 466, 1912.

42. Hill, A.V., " The position occupied by the production of heat in the chain of processes contributing to muscular contraction ", Journal of Physiology, 42: 1-14, 1911.

43. Hill, A.V., Long, C.N.H., and Lupton, H., " Muscular exercise, lactic acid, and the supply and utilization of oxygen ", Proceedings of the Royal Society Series B, 96, 97, 98, 99: 436-74, 84-137, 65-76, 156-66, 1928.

44. Hill, A.V., " The diffusion of lactic acid and oxygen through tissues ", Proceedings of the Royal Society, Series B, 104: 39-96, 1929.

45. Huckabee, W.E., " Relationships of pyruvate and lactate during anaerobic metabolism ", Journal of Clinical Investigation, 37: 255-63, 1958.

46. Jervell, Otto, " Investigation of the concentration of lactic acid in blood and urine ", Acta Medica Scandinavica, suppl 24, 1928.

47. Keul, J., D. Keppler and E. Doll, " Lactic-pyruvate ratio and its relation to oxygen pressure in arterial, coronarvenous, and femoral venous blood ", Arch. Inter. Physiologie and Biochemie, 75: Fasc 4, 1967.

48. Keul, J., E. Doll and D. Keppler, " The substrate supply of the human skeletal muscle at rest, during and after work ", Experientia, 23: 1-6, 1967.

49. Knuttgen, H.G., " Oxygen debt, lactate, pyruvate, and excess lactate after muscular work ", Journal of Applied Physiology, 17: 639-44, 1962.

50. Kwanji, Tsuji, " Lactic acid metabolism in the isolated heart ", Journal of Physiology, 50: 311-21, 1915-16.

51. Lehninger, Albert L., " Molecular basis of mitochondrial structure and function ", Molecular Organization and Biological Function, Harper and Row, Inc., New York, 107-27, 1967.

52. Levy, Matthew N., " Lactate uptake by intact kidney " Annals of the New York Academy of Sciences, 199: art 3, 1029-37, 1965.

53. Liljestrand, S.H., Willson, W.D., " The excretion of lactic acid in urine after muscular exercise ", Journal of Biological Chemistry, 65: 773-82, 1925.

54. Lombard, Warren P., " Some influences which affect the power of voluntary contraction ", Journal of Physiology, 13: 1-58, 1892.

55. Lowenstein, J.M., " The regulation of carbohydrate utilization ", Oxygen in The Animal Organism, MacMillan Co., New York, 163-77, 1964.

56. Margaria, R., Cerretelli P., and Mangili F., " Balance and kinetics of anaerobic energy release during strenuous exercise in man ", Journal of Applied Physiology, 19: #4 623-28, 1964.

57. Margaria, R., Edwards, H.T., and Dill D.B., " The possible mechanisms of contracting and paying the oxygen debt and the role of lactic acid in muscular contraction ", American Journal of Physiology, 106: 689-715, 1933.

58. Margaria, R., " Kinetics and mechanism of oxygen debt in man ", Journal of Applied Physiology, 18: 1963.

59. Margaria, R., Edwards, H.T., " The removal of lactic acid from the body during recovery from fatigue ", American Journal of Applied Physiology, 107: 681-86, 1934.

60. Margaria, R., Edwards, H.T., " The sources of energy in muscular work performed in anaerobic conditions ", American Journal of Physiology, 108: 341-48, 1934.

61. Mawson, C.H., " The lactic acid metabolism of frogs muscle poisoned with iodo acetic acid ", Journal of Physiology, 75: 200-212, 1932.

62. Olsen, R.E., " Excess lactate and anaerobiosis ", Annals of Internal Medicine, 59: 960-63, 1963.

63. Owles, Harding W., " Alterations in the lactic acid content of the blood as a result of light exercise, and the associated changes in the $CO_2$ combining power of the blood and the alveolar $CO_2$ pressure ", Journal of Physiology, 69: 214-37, 1930.

64. Pernow, B., John Wahren and Staffan Zetterquist, " Studies of the peripheral circulation and metabolism in man ", Acta Physiologica Scandinavica, 64: 289-98, 1965.

65. Piha, R.S., " Augmentation of pyruvic acid and deficiency of oxygen supply ", Acta Physiologica Scandinavica, 44: suppl 151, 32-35, 1958.

66. Ransom, F., " The action of caffiene on muscle ", Journal of Physiology, 42: 144-55, 1911.

67. Raper, H.S., " On the formation of fatty acids from lactic acid when fused with caustic alkalies " , Journal of Physiology, 32: 216-20, 1905-06.

68. Rowell, L.B., Kenneth K Kraning II, Thomas O. Evans, J. Ward Kennedy, J.R. Blackman, and Fusako Kusumi, " Splanchnic removal of lactate and pyruvate during prolonged exercise in man " , Journal of Applied Physiology, 21: #6, 1773-83, 1966.

69. Ryffle, J.H., " Experiments on oactic acid formation in man ", Journal of Physiology, 39: xxix, 1909-10.

70. Saiki, H., R. Margaria and F. Cuttica, " Lactic acid production in submaximal work ", Int. Zangnew Physiol. einschl Arbeitsphysiol, 24: 57-61, 1967.

71. Saktor, B., Dick, A.R., " Oxidation of extramitochondrial NADH by various tissues of the mouse ", Science, 145: 606-07, 1964.

72. Sigma Chemical Company, " The determination of L+ lactic acid in blood and other fluids at 340 mu. ", Technical Bulletin # 825, 44, St. Louis, Mo., 1965.

73. Sigma Chemical Co., " Enzymatic determination of pyruvic acid in blood at 340 mu. ", Technical Bulletin # 725, St. Louis, Mo., 1966.

74. Stainsby, W.N., and Welch, H.G., " Lactate metabolism of contracting dog skeletal muscle in situ ", American Journal of Physiology, 211: 177-83, 1966.

75. Strandell, T., " Heart rate, arterial lactate concentration and oxygen uptake during exercise in old men compared to young men ", Acta Physiol Scandinavica, 60: 197-206, 1964.

76. Tipton, C.M., and Tharp, G.D., " Influence of training on tissue respiration ", Int. Zangew Physiol, 21: 236-39, 1966.

77. Warnock, L.G., R.E. Koeppe, N.F. Inciardi, and W.E. Wilson, " L+ and D- lactate as precusors of muscle glycogen " Annals of the New York Academy of Sciences, 119: art 3, 1048-60, 1965.

78. Wasserman, K., George G. Burton, and Antonius L. Van Kessel, " Excess lactate concept and oxygen debt of exercise ", Journal of Applied Physiology, 20: #6 1299-06, 1965.

79. Weber, G., Lea, M.A., Hird Convery, H.J., and Stamm, N.B., " Regulation of gluconeogenisis and glycolysis, studies on mechanisms of controlling enzyme activity ", Advances in Enzyme Regulation 5: Permagon Press Inc., New York, 257-99, 1967.

80. Weil, Rudolf, Peh-Ping Ho, and Norman Altszuler, " Effect of FFA on metabolism of pyruvic and lactic acids ", American Journal of Physiology, 208: #5, 887-91, 1965.

81. White, A., Handler, P., and E.L.Smith, Principles of Biochemistry, 3rd edition, McGraw-Hill, New York, 1964.

82. Williams, C.G., A.J. N. DuRaan, M.J. VonRahden, and C.H. Wyndham, " The capacity for endurance work in highly trained men ", Int. Zangew Physiol, 26: 141-49, 1968.

83. Winer, B.J., Statistical Principles in Experimental Design, McGraw-Hill, New York, 1962.

# APPENDIX A

## CONSENT FORM

UNIVERSITY OF ALBERTA

FACULTY OF PHYSICAL EDUCATION

CONSENT TO PARTICIPATE IN A RESEARCH STUDY

SUBJECT.....DATE.....TIME A.M. / P.M.

1. I agree to participate in an investigation and in relation to this hereby authorize Drs. .....and/or such assistants as may be selected by them, to perform the following procedure (s):

.....

.....

.....

2. Drs. .....have explained the purpose of this study and I understand the routine of the procedure outlined above.

..... .....
Witness Signature of subject

If the subject is unable to sign or is under 18 years of age, complete the following:

The subject is a minor (.....years of age ).

or

The subject is unable to sign because.....

As the closest relative or legal guardian I hereby sign on his/her behalf:

.....
Witness Signature Relationship

# APPENDIX B

## STATISTICAL SUMMARY TABLES

## AND RAW DATA

TABLE 18

LACTATE SUMMARY TABLES

SUMMARY TABLE AB

| | working | nonworking | totals |
|---|---|---|---|
| trained | 663.6* | 482.1 | 1145.7 |
| semi-trained | 674.1 | 567.7 | 1241.8 |
| sedentary | 494.8 | 364.9 | 859.7 |
| totals-- | 1832.5 | 1414.7 | 3247.2 |

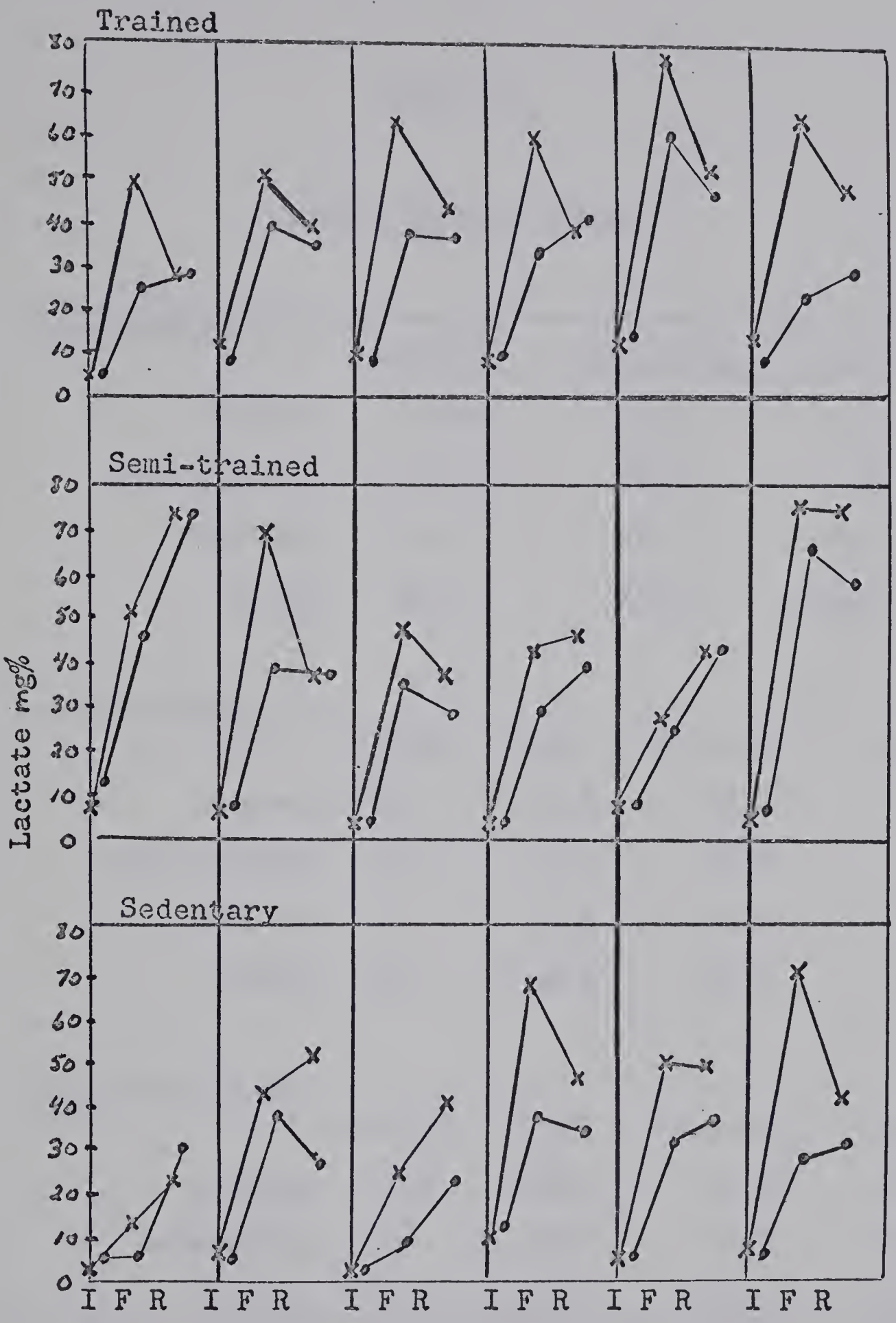
SUMMARY TABLE AC

| | Initial | Final | Recovery | totals |
|---|---|---|---|---|
| trained | 105.3 | 581.4 | 459.0 | 1145.7 |
| semi-trained | 98.7 | 550.9 | 592.2 | 1241.8 |
| sedentary | 76.0 | 394.8 | 388.8 | 859.7 |
| totals-- | 280.0 | 1527.1 | 1440.0 | 3247.2 |

SUMMARY TABLE BC

| | Initial | Final | Recovery | totals |
|---|---|---|---|---|
| working | 136.9 | 926.2 | 769.3 | 1832.5 |
| nonworking | 143.1 | 600.9 | 670.7 | 1414.7 |
| totals | 280.0 | 1527.1 | 1440.0 | 3247.2 |

* total lactate mg%




I- initial, F- final, R- recovery

x- working sample, o - nonworking sample

FIGURE 19

LACTATE ACTIVITY DURING MAXIMAL EXERCISE

TABLE 19

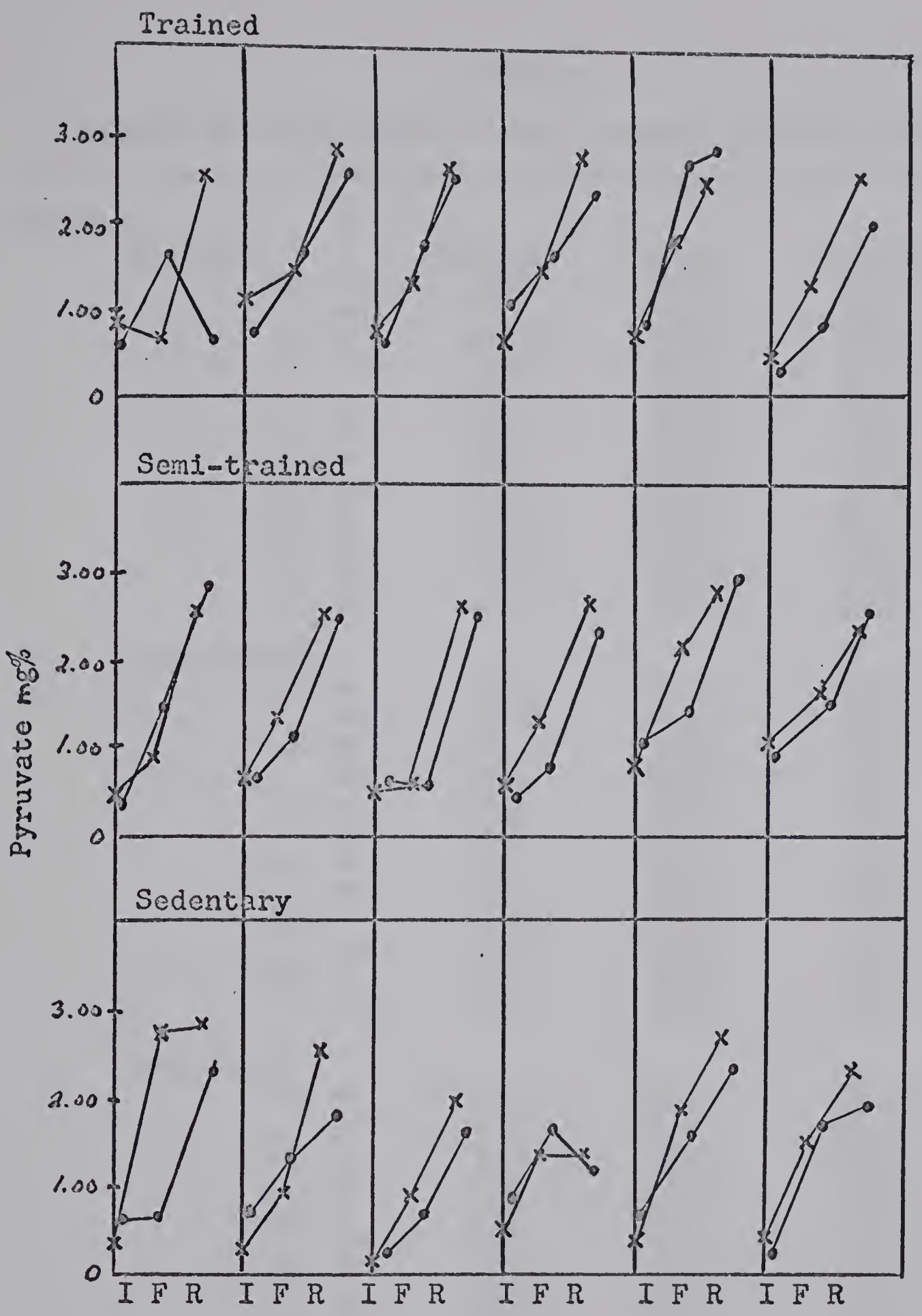
PYRUVATE SUMMARY TABLES

| SUMMARY TABLE AB | working | nonworking | totals |
|---|---|---|---|
| trained | 28.8* | 27.3 | 56.1 |
| semi-trained | 24.7 | 28.8 | 53.5 |
| sedentary | 21.1 | 19.2 | 40.4 |
| totals | 74.7 | 75.3 | 150.0 |

| SUMMARY TABLE AC | Initial | Final | Recovery | totals |
|---|---|---|---|---|
| trained | 9.2 | 17.8 | 29.0 | 56.1 |
| semi-trained | 7.6 | 15.0 | 30.9 | 53.5 |
| sedentary | 5.1 | 13.7 | 21.7 | 40.4 |
| totals | 21.9 | 46.5 | 81.6 | 150.0 |

| SUMMARY TABLE BC | Initial | Final | Recovery | totals |
|---|---|---|---|---|
| working | 12.1 | 20.7 | 41.8 | 74.7 |
| nonworking | 9.8 | 25.7 | 39.7 | 75.3 |
| totals | 21.9 | 46.5 | 81.6 | 150.0 |

* total pyruvate mg%




I- initial, F- final, R- recovery

x- working sample, o - nonworking sample

FIGURE 20

PYRUVATE ACTIVITY DURING MAXIMAL EXERCISE

TABLE 20

LACTATE CONCENTRATIONS ( mg% ) DURING MAXIMAL EXERCISE

| Group: | | Initial | Final | Recovery |
|---|---|---|---|---|
| Trained | | | | |
| #1. | w | 5.2 | 50.1 | 29.4 |
| | nw | 5.3 | 26.3 | 29.3 |
| 2. | w | 10.2 | 51.2 | 38.7 |
| | nw | 7.6 | 38.7 | 34.6 |
| 3. | w | 9.1 | 60.3 | 41.0 |
| | nw | 8.3 | 36.5 | 35.5 |
| 4. | w | 7.3 | 58.9 | 37.2 |
| | nw | 8.8 | 33.1 | 39.1 |
| 5. | w | 11.4 | 77.6 | 51.6 |
| | nw | 13.4 | 61.1 | 46.0 |
| 6. | w | 11.7 | 64.0 | 48.2 |
| | nw | 6.5 | 23.6 | 28.4 |
| Semi-trained | | | | |
| 7. | w | 8.8 | 53.5 | 75.7 |
| | nw | 14.4 | 45.7 | 75.7 |
| 8. | w | 7.3 | 69.1 | 36.6 |
| | nw | 9.2 | 39.1 | 36.8 |
| 9. | w | 5.7 | 47.1 | 37.8 |
| | nw | 4.6 | 34.6 | 26.5 |
| 10. | w | 4.8 | 42.1 | 44.9 |
| | nw | 5.2 | 28.1 | 39.2 |
| 11. | w | 11.5 | 26.3 | 42.0 |
| | nw | 11.3 | 23.6 | 43.2 |
| 12. | w | 7.1 | 77.0 | 76.5 |
| | nw | 8.8 | 64.4 | 57.3 |
| Sedentary | | | | |
| 13. | w | 2.7 | 13.4 | 23.9 |
| | nw | 6.6 | 5.9 | 31.9 |
| 14. | w | 6.2 | 42.7 | 50.6 |
| | nw | 5.3 | 37.2 | 26.1 |
| 15. | w | 3.6 | 24.2 | 40.2 |
| | nw | 3.6 | 9.1 | 22.2 |
| 16. | w | 10.7 | 67.0 | 44.6 |
| | nw | 12.7 | 36.4 | 33.4 |
| 17. | w | 6.0 | 51.9 | 50.3 |
| | nw | 6.5 | 32.0 | 36.9 |
| 18. | w | 7.1 | 69.5 | 40.1 |
| | nw | 5.0 | 25.5 | 28.6 |

w - working limb
nw - nonworking limb

TABLE 21

PYRUVATE CONCENTRATIONS ( mg% ) DURING MAXIMAL EXERCISE

| Group: | | Initial | Final | Recovery |
|---|---|---|---|---|
| Trained | | | | |
| #1. | w | .98 | .88 | 2.52 |
| | nw | .65 | 1.56 | .87 |
| 2. | w | 1.09 | 1.40 | 2.82 |
| | nw | .67 | 1.56 | 2.63 |
| 3. | w | .84 | 1.33 | 2.65 |
| | nw | .75 | 1.63 | 2.50 |
| 4. | w | .73 | 1.40 | 2.88 |
| | nw | 1.03 | 1.47 | 2.35 |
| 5. | w | .77 | 1.73 | 2.41 |
| | nw | .82 | 2.74 | 2.80 |
| 6. | w | .52 | 1.25 | 2.61 |
| | nw | .39 | .85 | 2.00 |
| Semi-trained | | | | |
| 7. | w | .51 | .87 | 2.59 |
| | nw | .46 | 1.40 | 2.78 |
| 8. | w | .65 | 1.20 | 2.55 |
| | nw | .65 | 1.03 | 2.54 |
| 9. | w | .51 | .52 | 2.67 |
| | nw | .56 | .53 | 2.53 |
| 10. | w | .53 | 1.11 | 2.70 |
| | nw | .42 | .61 | 2.30 |
| 11. | w | .66 | 2.03 | 2.67 |
| | nw | .94 | 1.31 | 2.77 |
| 12. | w | 1.03 | 1.47 | 2.12 |
| | nw | .91 | 1.30 | 2.33 |
| Sedentary | | | | |
| 13. | w | .41 | 2.84 | 2.88 |
| | nw | .59 | .59 | 2.28 |
| 14. | w | .35 | .84 | 2.66 |
| | nw | .67 | 1.20 | 1.74 |
| 15. | w | .20 | .96 | 1.95 |
| | nw | .23 | .59 | 1.58 |
| 16. | w | .49 | 1.36 | 1.37 |
| | nw | .88 | 1.47 | 1.07 |
| 17. | w | .42 | 1.91 | 2.60 |
| | nw | .62 | 1.61 | 2.30 |
| 18. | w | .44 | 1.50 | 2.19 |
| | nw | .20 | 1.65 | 1.93 |

w - working limb
nw - nonworking limb

TABLE 22

LACTATE CONCENTRATIONS ( mg% ) DURING SUBMAXIMAL EXERCISE

| Group: | | | Initial | 20m | 40m | 60m | 80m | 100m | 120m | Final | Recovery* |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Trained | #1. | w | 4.0 | 8.6 | 6.9 | 6.9 | - | - | - | 23.7 | 11.6 |
| | | nw | 11.4 | 8.0 | 5.0 | 11.4 | - | - | - | 27.2 | 7.1 |
| | 2. | w | 7.7 | 40.4 | 43.0 | - | - | - | - | 45.6 | 17.6 |
| | | nw | 6.9 | 38.7 | 30.6 | - | - | - | - | 30.1 | 18.0 |
| | 3. | w | 11.5 | 12.0 | 21.1 | 12.4 | 13.3 | 23.3 | 36.5 | 47.7 | 27.1 |
| | | nw | 11.4 | 12.3 | 16.8 | 13.9 | 9.9 | 21.9 | 43.7 | 36.1 | 28.6 |
| | 4. | w | 4.3 | 13.4 | 23.5 | - | - | - | - | 14.4 | 7.7 |
| | | nw | 6.5 | 19.8 | 15.3 | - | - | - | - | 15.9 | 8.3 |
| | 5. | w | 10.8 | 60.2 | 37.8 | - | - | - | - | 44.8 | 18.5 |
| | | nw | 11.1 | 54.0 | 31.0 | - | - | - | - | 32.6 | 27.9 |
| | 6. | w | 7.4 | 11.9 | 23.7 | 28.9 | - | - | - | 66.0 | 31.0 |
| | | nw | 7.0 | 14.2 | 27.5 | 25.1 | - | - | - | 81.0 | 43.9 |
| Semi-trained | 7. | w | 4.0 | 11.4 | 28.6 | - | - | - | - | 35.3 | 15.3 |
| | | nw | 4.6 | 14.5 | 14.2 | - | - | - | - | 23.1 | 12.4 |
| | 8. | w | 4.9 | 13.1 | 22.6 | 18.1 | - | - | - | 20.0 | 9.7 |
| | | nw | 6.5 | 12.0 | 25.7 | 16.9 | - | - | - | 12.9 | 9.6 |
| | 9. | w | 3.8 | 9.5 | 13.2 | - | - | - | - | 54.1 | 15.2 |
| | | nw | 3.6 | 9.6 | 8.0 | - | - | - | - | 25.3 | 17.0 |
| | 10. | w | 7.1 | - | - | - | - | - | - | 36.8 | 28.6 |
| | | nw | 9.3 | - | - | - | - | - | - | 35.5 | 27.3 |
| | 11. | w | 10.8 | 14.3 | 16.2 | 15.4 | - | - | - | 18.2 | 10.2 |
| | | nw | 8.1 | 21.2 | 14.3 | 13.1 | - | - | - | 15.9 | 12.0 |
| | 12. | w | 10.9 | - | - | - | - | - | - | 79.1 | 96.3 |
| | | nw | 11.4 | - | - | - | - | - | - | 73.5 | 43.0 |
| Sedentary | 13. | w | 8.4 | 13.8 | 14.2 | - | - | - | - | 13.3 | 8.9 |
| | | nw | 5.8 | 21.3 | 13.8 | - | - | - | - | 16.9 | 10.2 |
| | 14. | w | 5.5 | 12.3 | - | - | - | - | - | 30.4 | 16.2 |
| | | nw | 5.3 | 10.1 | - | - | - | - | - | 25.2 | 20.3 |
| | 15. | w | 4.8 | 18.9 | - | - | - | - | - | 21.8 | 8.9 |
| | | nw | 7.5 | 17.2 | - | - | - | - | - | 16.4 | 14.4 |
| | 16. | w | 5.4 | 7.6 | 14.5 | - | - | - | - | 44.0 | 14.3 |
| | | nw | 5.1 | 9.1 | 11.8 | - | - | - | - | 29.0 | 38.5 |
| | 17. | w | 4.6 | 28.2 | 18.1 | - | - | - | - | 12.0 | 20.3 |
| | | nw | 6.5 | 12.9 | 13.6 | - | - | - | - | 18.7 | 23.6 |
| | 18. | w | 4.1 | 49.7 | - | - | - | - | - | 20.8 | 15.3 |
| | | nw | 3.7 | 44.2 | - | - | - | - | - | 26.5 | 16.7 |

* 10 minutes following fatigue; m= minutes

TABLE 23

PYRUVATE CONCENTRATIONS ( mg% ) DURING SUBMAXIMAL EXERCISE

| Group: | | Initial | 20m | 40m | 60m | 80m | 100m | 120m | Final | Recovery* |
|---|---|---|---|---|---|---|---|---|---|---|
| Trained | #1. w | .28 | 1.35 | .92 | 1.09 | - | - | - | 1.98 | 1.28 |
| | nw | .30 | .70 | .60 | .81 | - | - | - | 1.46 | 1.08 |
| | 2. w | .62 | 2.55 | 2.57 | - | - | - | - | 2.63 | 1.28 |
| | nw | .60 | 1.48 | 1.35 | - | - | - | - | 1.59 | .95 |
| | 3. w | .40 | .63 | .90 | .58 | .46 | 1.30 | 1.18 | 1.54 | 1.04 |
| | nw | .52 | .58 | .70 | .47 | .14 | .94 | 1.64 | 1.08 | 1.10 |
| | 4. w | .43 | .78 | 1.36 | - | - | - | - | 1.65 | .45 |
| | nw | .53 | .96 | .92 | - | - | - | - | .70 | .94 |
| | 5. w | .69 | 1.94 | 1.28 | - | - | - | - | 1.29 | 1.04 |
| | nw | .69 | 1.51 | 1.09 | - | - | - | - | 1.52 | 1.01 |
| | 6. w | .71 | .94 | 1.08 | 1.66 | - | - | - | 1.75 | 1.97 |
| | nw | .60 | .70 | 1.55 | 1.50 | - | - | - | 2.50 | 1.82 |
| Semi-trained | 7. w | .31 | .90 | 1.75 | - | - | - | - | 1.32 | .71 |
| | nw | .52 | .69 | 1.22 | - | - | - | - | 1.41 | 1.02 |
| | 8. w | .33 | .64 | 1.16 | 1.52 | - | - | - | .89 | .41 |
| | nw | .41 | .93 | .58 | .73 | - | - | - | .26 | .77 |
| | 9. w | .24 | .86 | 1.00 | - | - | - | - | 1.62 | 1.97 |
| | nw | .56 | .69 | .45 | - | - | - | - | .60 | 1.52 |
| | 10. w | .46 | - | - | - | - | - | - | 1.72 | 1.06 |
| | nw | .57 | - | - | - | - | - | - | 1.32 | .74 |
| | 11. w | .58 | - | - | - | - | - | - | 1.72 | 1.51 |
| | nw | .31 | - | - | - | - | - | - | 2.77 | 1.90 |
| | 12. w | .37 | 1.08 | 1.18 | 1.15 | - | - | - | .68 | .68 |
| | nw | .45 | .88 | .74 | .83 | - | - | - | .75 | .83 |
| Sedentary | 13. w | .43 | 1.00 | .82 | - | - | - | - | .62 | .23 |
| | nw | .25 | 1.02 | .75 | - | - | - | - | 1.34 | .42 |
| | 14. w | .81 | .23 | - | - | - | - | - | 1.51 | 1.04 |
| | nw | .71 | .25 | - | - | - | - | - | 1.20 | 1.18 |
| | 15. w | .17 | 1.16 | - | - | - | - | - | 1.17 | .95 |
| | nw | .37 | 1.12 | - | - | - | - | - | 1.16 | 1.70 |
| | 16. w | .90 | 1.08 | .60 | - | - | - | - | 2.07 | 1.08 |
| | nw | .70 | .54 | .23 | - | - | - | - | 1.63 | 1.23 |
| | 17. w | .60 | .17 | 1.27 | - | - | - | - | 1.60 | .87 |
| | nw | .81 | .90 | .97 | - | - | - | - | .65 | 1.38 |
| | 18. w | .05 | 1.96 | - | - | - | - | - | .24 | 1.21 |
| | nw | .12 | 2.25 | - | - | - | - | - | 1.40 | .69 |

* 10 minutes following fatigue; m= minutes